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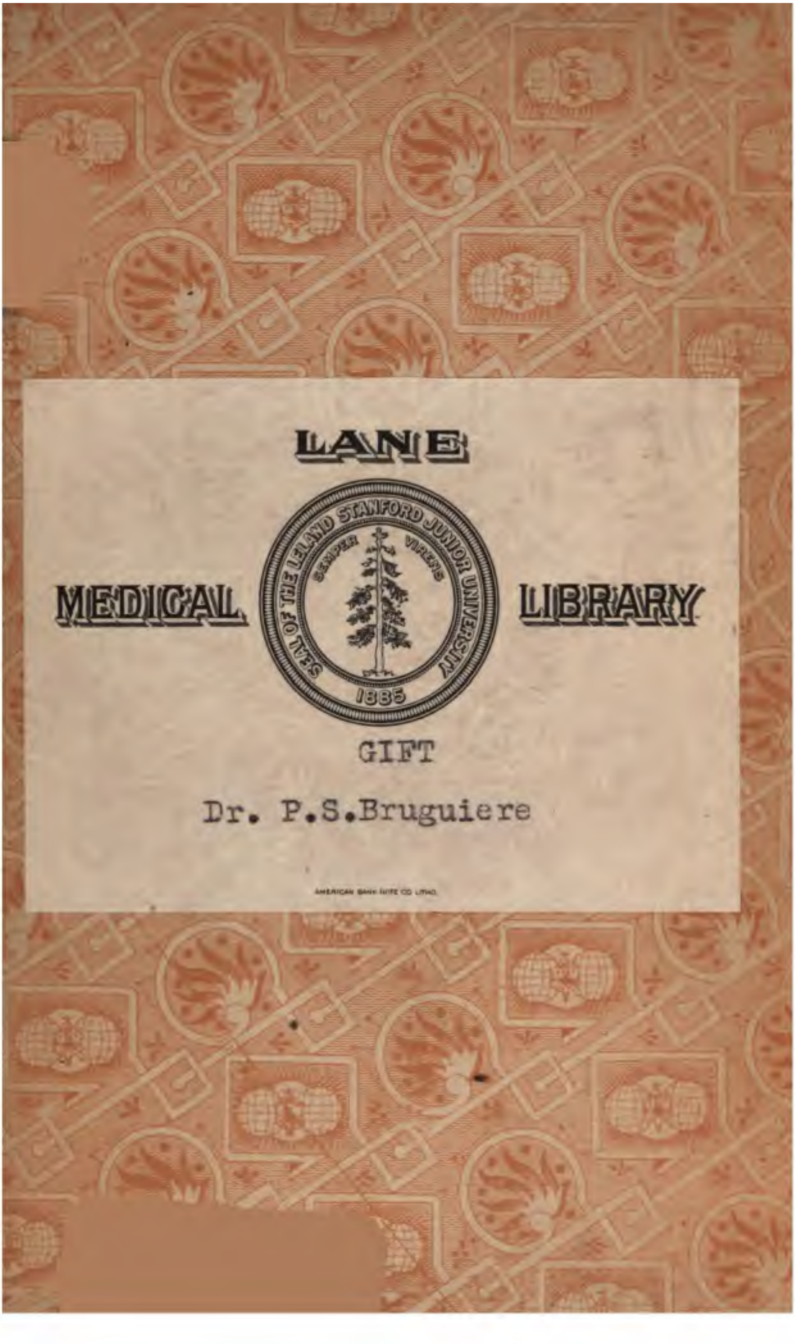

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PREFACE.

THIS volume comprises a part of the topics lectured upon by me for several years in the Chicago Medical College. They have been elaborated from my lecture notes. It has been my endeavor to describe as clearly, concisely, and fully as possible the subjects of this book. I have avoided controversial topics, and may sometimes have erred by stating positively what is rather probably than positively true. I have tried to make the subject of treatment especially full, and have endeavored to give explicit directions as to the time when individual drugs should be used, the exact indications for them, and their mode of action in each disease. In order to prevent repetitions when the mode of action of drugs was the same in several allied diseases this fact is stated, and the details of action must be learned from the description of the first of the allied maladies.

That the volume might not outgrow the "Ready Reference Series" for which it was intended, numerous foot-notes and tables of bibliography have not been added to the text.

N. S. DAVIS, JR.

65 RANDOLPH ST., CHICAGO,
October, 1892.

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SECTION I.

Diseases of the Bronchi, Lungs, and Pleura.

DISEASES OF THE BRONCHI.

CHAPTER I.

ASTHMA.

Nature.—Asthma is an expiratory dyspnœa which occurs paroxysmally, and is usually sudden in its onset. As a rule, the paroxysms are, at longest, of only a few hours' duration. The exact nature of these attacks is unknown. By most clinicians they are believed to be due to spasmodic contraction of the bronchi, which is excited through the agency of the nervous system. A smaller number believe that the narrowing of the bronchi is due to a sudden and very great congestion of their mucous membranes. Such a congestion could only be produced through the active agency of the vasomotor nervous system. It may be likened to the cutaneous congestion and swelling of hives. Intense congestion of the trachea and that part of the right bronchus which can be seen in a laryngeal mirror can be observed during an attack of asthma. A still smaller number of observers explain the paroxysm upon the supposition that it is due to a spasm of the diaphragm, which causes an enlargement of the thorax, and, therefore, sudden dilatation of the lungs and difficult and unnatural respiration. This view is based upon the fact that in many cases of asthma no movement of the diaphragm can be demonstrated. There are other cases, however, in which movements can be demonstrated. The explanation is not, therefore, of universal applicability. That muscular contraction of the bronchi can be produced has been demonstrated by physiologists. The suddenness of the onset of these attacks, and often of their cessation, as well as the prompt relief so frequently obtained from such drugs as chloroform and chloral, demonstrates the dominant influence of the nervous system in their production. Most, if not all, cases of asthma are of reflex origin. Therefore, the physiological mechanisms which are involved consist of (1) a source of irritation or sensitive nerve-endings that are subject to irritation; (2) the central nervous system by which the irritation experienced by the sensitive nerves is reflected to the motor ones; (3) the motor nerves and muscular fibres of the bronchi which constitute the focus of irritation. Undoubtedly the mucous membranes become reddened simultaneously. A temporary expansion of the lung-alveoli in this disease is the result of overfilling, because of imperfect emptying during expiration. The calibre of the smaller bronchi is diminished; and, as inspiration, which is accomplished purely by muscular action, is a much more forceful act than expiration, which is brought about, chiefly by the elasticity of the lung-tissue, the weight of the thoracic and abdominal walls, and in dyspnœa the exercise of voluntary muscles, which do not, however, act under the most advantageous conditions, more air gradually enters the alveoli than can be forced out. This temporary expansion of the lungs leads to an enlargement of the entire thorax during the dyspnœic paroxysm.

Symptoms.—The asthmatic attacks usually occur in periods lasting from a few days to several weeks, during which time they recur at regular, and usually at daily, intervals. The periods may be weeks or months apart. Less frequently a single paroxysm of dyspnœa will occur not followed by others, or followed by them only after a long intermission.

In the majority of cases there are no premonitory symptoms, but in this respect, as in the causation of the disease, there is much of idiosyncrasy in each case. In a proportion of them there are premonitory symptoms that are peculiar to each individual. A person is occasionally found whose attacks are uniformly preceded by an unnatural drowsiness, or it may be by sneezing, or itchiness, or flatulence, or by the passage of large quantities of very pale urine, or by various other symptoms which the sufferer's experience leads him to recognize as warnings of the approach of an attack.

The paroxysm of dyspnœa almost invariably occurs in the earliest morning hours, and in a majority of cases between two and four. The sufferer usually awakes from a sound sleep with a feeling of oppression in breathing. Almost at once the dyspnœa becomes intense. If the patient is not accustomed to the attacks relief is usually sought at the open window; those who have often experienced the sufferings of asthma assume at once some favorite attitude which they have learned makes it possible for them to breathe with the most ease. These attitudes are various, but their object uniformly is to fix the shoulders rigidly so that the unusual muscles of respiration which find origin about them can act most advantageously upon the thorax. A favorite position with many is a sitting one upon the edge of the bed or upon a chair, the sides of which are grasped by the straightened arms, which thus hold the shoulders rigid. The body is bent a little forward, the head is thrown back to straighten the neck, and the mouth is opened to permit the freest ventilation of the lungs. The countenance expresses at first anxiety, and later extreme distress, as the symptoms of suffocation intensify. The violent muscular exertion that the struggle for breath involves produces at first a warm, moist perspiration over the upper part of the body and face. As the dyspnœa is prolonged and intensified the skin becomes cool and clammy; in color it is often ashen and the lips and finger-nails become purplish. In a word, marked cyanosis is developed. The pulse is quick and frequently irregular, small, and rigid. The veins of the neck are unusually full and may stand out like whipcords. This is due to the interference with normal venous circulation by the change in intra-thoracic pressure caused by the dyspnœic breathing. The bodily temperature is rarely abnormal.

A physical examination shows that, while the respiratory movements are so labored, they are no more or very little more rapid than normal. The thorax is dilated, and it retains constantly the normal inspiratory position. The intercostal spaces do not change in width with the respiratory movements, but are persistently stretched to their widest extent. The ribs do not move freely on their axes with each respiration. As the chest is thus constantly extended to its utmost, inspiration is only effected by lifting the thorax as a whole with the unusual muscles of respiration. The inspiratory act is short and jerky in character and the expiratory is very much prolonged and labored. During inspiration the lower part of the chest will be observed, in children, not to be expanded, but to be retracted. This appearance is little noticed in adults, as their ribs are too rigid to be thus bent, but the lower intercostal spaces, as well as often the supra-clavicular spaces, are retracted during inspiration. This is due to the low pressure within the thorax during inspiration. The

air is not able to enter and dilate the lungs when the thorax is lifted; therefore, the atmosphere's weight presses in the yielding parts. Palpation usually reveals no change, although occasionally a bronchial fremitus can be felt. Percussion reveals an increased resonance, which is uniform upon both sides of the chest. The area of resonance is increased. The area of dullness over the heart is diminished by the distension of the overlapping portion of the lung, and the area of liver-dullness is depressed and seen to change less in its horizon with inspiration and expiration than natural. These changes are due to the unusual distension of the lungs and imperfect respiration maintained by them. The apex-beat of the heart can frequently not be seen or felt, and its sounds are somewhat distant because of the overlapping lung. Auscultation is not necessary in order to hear the abnormal respiratory sounds, for they are so loud that they can be heard many feet from the sufferer. If the ear is placed on the chest, vesicular sounds will be found entirely absent, and only piping and crowing will be audible, and the latter can be heard as readily by the by-stander. The inspiratory sound is very short, the expiratory much prolonged.

Usually when the dyspnœa is most intense, the cyanosis most marked, and fatal suffocation apparently imminent, relief comes and the oppression rapidly abates. With or very shortly preceding this abatement a slight cough begins. In many cases it is so slight as to be unnoticed. Usually it is accompanied by the expectoration of a small number of sputa-chunks, of small size and glary, gray, adhesive character. If the sputa is examined microscopically it is found, in a large proportion of cases, to contain characteristic elements. The most striking are spiral coils of thread-like fibrils. They are not strictly peculiar to asthma, although they occur with it more uniformly than with any other disease. They have been noticed in the sputa of capillary bronchitis and rarely in that of croupous pneumonia and phthisis. The needle-like crystals of Charcot can also be frequently observed. The cellular elements are those usual to sputa, and are not peculiar to the disease.

The paroxysm that I have described typifies those of the severest type of the disease. Every grade of milder form can be observed, even to that in which there is little disturbance of respiration more than a feeling of oppression. The duration of the paroxysm of dyspnœa is variable. It may last a few minutes only,—usually it persists for from one to three hours. In rarer cases it will last for several days with little change, except an intensification during the night. These are usually cases in which bronchitis is present, and is the exciting cause of the asthma. Such cases are accompanied by more or less persistent cough and somewhat accelerated breathing. They can be distinguished from capillary bronchitis with difficulty, except by the paroxysmal character of the dyspnœa and by the absence of fever.

The intervals between attacks of frankly-spasmodic asthma are passed by the patient in perfect comfort. When the dyspnœa first ceases the sufferer is so exhausted by the preceding laborious breathing that almost invariably he drops upon the bed and falls into a quiet and sometimes protracted sleep. On awakening from this sleep the patient usually feels perfectly comfortable. At this time an examination will reveal no abnormal physical signs. In most instances the dyspnœa will recur the following night, and may for several more.

Diagnosis.—Diagnosis is usually not difficult; the sudden onset, the severity of the dyspnœa, the sudden cessation, the complete restoration during the intervals, and repetition of the attacks are characteristic. It is necessary, at times, to distinguish the dyspnœa of spasmodic asthma from that produced by laryngeal or tracheal obstruction. In the latter cases the dyspnœa is mainly inspiratory, while in asthma it is expiratory. Upon search a cause for the dyspnœa is found in the upper air-passages. The noise of respiration is loudest about the throat.

From capillary bronchitis we can differentiate spasmodic asthma: 1. By the mode of onset, which, in the former, is somewhat slow and gradual, not sudden. 2. By the absence of fever in asthma. It must be remembered, however, that an apyretic capillary bronchitis occurs at times. 3. By the hurried respiration of the bronchitis. 4. By the presence of cough. 5. By the sudden cessation of the dyspnœa of asthma and more persistent character of that of the inflammatory affection. From emphysema it is to be distinguished (1) by the chronicity of the dyspnœa, (2) by its lack of paroxysmal character, and (3) by the permanent distension of the chest in the former. From cardiac asthma (1) by the existence of a cardiac lesion that will explain its cause, (2) by its persistence, and (3) by the absence of a history of attacks prior to the development of the cardiac trouble.

It must be remembered that bronchitis, emphysema, and cardiac disease may be complicated by true spasmodic asthma. In all cases of persistent dyspnœa with nightly exacerbations, some other cause than simply spasm of the bronchi may be quite confidently suspected, even if it cannot be proven.

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It must be remembered that bronchitis, emphysema, and cardiac disease may be complicated by true spasmodic asthma. In all cases of persistent dyspnœa with nightly exacerbations, some other cause than simply spasm of the bronchi may be quite confidently suspected, even if it cannot be proven.

ease is only obtainable by either removing the cause of the disease from the sufferer or removing the sufferer from the cause. The latter method is especially applicable to the cases of hay fever in which foreign bodies in the atmosphere are the exciting cause and the nasal mucous membrane the source of irritation of the disease and complicating asthma. A change of climate and, therefore, of air is curative. The localities in this country that afford most perfect exemption are the White Mountains, Mackinac, many localities along the shore of Lake Superior, and numerous places in the more elevated parts of the Rocky and other mountainous regions. A residence in the heart of a thickly-populated city will often grant to individual cases immunity, although they may suffer severely in neighboring suburbs. As these attacks are most likely to occur at certain seasons, especially in August and September, and less frequently in June, temporary changes of abode at these times will usually give exemption to those who are liable to the attacks. Many of the afflicted cannot take advantage of such prophylactic treatment.

To prevent asthmas that result from the existence of chronic inflammation or tumors within the nostrils, a destruction of the irritating tissue must be effected. A temporary relief can often be obtained by the use of local anæsthetics. Rarely, the source of irritation is found in the pharynx or larynx. In such cases the irritant is usually a morbid growth or a chronic inflammation with hypertrophy. Such lesions must be treated just as are their analogues in the nasal cavity.

The indications for treatment are (*a*) to prevent the development of dyspnœa and (*b*) to relieve the dyspnœa when developed. In the intervals between the dyspnœic attacks the iodides are often prescribed, with marked benefit. Unfortunately, they do not uniformly ward off or mitigate the paroxysms. The cases in which good results are most uniformly obtained from their employment complicate chronic bronchitis. It is probable that their good effects are largely due to the property, which they possess, of promoting re-absorption of cellular exudates in inflamed tissues. The iodide of soda is the most eligible preparation for persistent employment. It should be given for weeks, and often for months. I have seen several cases apparently exempted from severe attacks by senecio aureus. The drug was not given during dyspnœa, but while the paroxysms were threatening, and at a season when the patients were usually afflicted by them. By the continued use of it for several weeks an actual outbreak was avoided.

An analysis of the mode of action of the drugs that are most successful in asthma shows that in one of three ways they relieve the spasm. We may, therefore, place them in three groups. The first includes those that affect the source of irritation, the second those that benumb the nerve-centre or reflector of irritation, and the third those that act upon the focus of irritation.

In the first group we must place a very promiscuous collection of drugs, since the source of irritation may be in almost any part of the body. We find, therefore, in this list, those medicines that allay irritability of nasal, pharyngeal, bronchial, and gastric mucous membranes, and also those that allay irritability of the womb and some of the parenchymatous organs.

In asthma of nasal origin there is necessary for its production not only the specific irritant in the atmosphere, but a peculiar sensitiveness of the nerve-endings which constitute the source of irritation, and possibly, also, of the nerve-centres. Advantage can be taken of these facts in mitigating and preventing the disease when a change to a pure, unirritating air is impossible. Thus, in hay fever, local anæsthetics applied to the nasal mucous membrane will frequently hold the disease in abeyance, or at least mitigate it. Of the remedial agents that can be topically applied for anæsthetic effects, cocaine is the most important. A 5- to 10- per-cent. solution may be sprayed into the nose through the anterior nares, and, when necessary, also applied to the posterior nares through the mouth. Or it can be employed by insufflating a powder composed of it and some bland diluent. A cocaine ointment may be used, a little being placed in the nostrils and allowed to melt and trickle backward, so as to anoint the mucous surfaces. This method is less efficacious than either of the others, since the drug is not applied so uniformly to all parts of the nose. It must be remembered, in regard to cocaine, that, if used in small amounts often, or in strong solution less frequently, symptoms of intoxication may be produced. I have rarely found it necessary to use preparations of more than 4-per-cent. strength. Often a few applications of cocaine will greatly aid in discovering the source of irritation, for cases occur in which we suspect the source to be in the nose or throat, and, if applications to these parts allay the dyspnœa, we may feel that our suspicions are well founded.

As a topical application morphia is also useful. It acts less promptly than cocaine, but often its effects are more lasting. A good formula consists of 4 per cent. of cocaine and 2 per cent. of morphia, mixed with some inert powder or with water, according as one wishes to make applications by insufflating or by spraying.

We must place in this miscellaneous group, also, the various expectorant and anodyne mixtures that are employed to allay laryngitis, trachitis, or bronchitis, since these inflammations are frequent causes of asthma, and, therefore, their cure will give exemption. The efficacy of such mixtures is greatly enhanced by combining with them drugs that belong to the second group, or those that allay the excitability of the reflex centres.

In the same way, asthma which accompanies uncompensated valvular disease of the heart is relieved by digitalis and similar drugs. They strengthen the heart's action and give greater tone to the blood-vessels, and thus reduce venous hyperæmia of the lungs and bronchi. These remedies accomplish more for such asthmatics than those that relax muscular spasm. They do good by stopping the irritation at its source. Cases in which the source of irritation is in the organs of the alimentary tract are relieved, and often permanently cured, by treatment of the primary lesions. Occasionally, a woman is found who is persistently troubled with asthma during pregnancy, although free from it at other times. Absolute relief is, so long as pregnancy lasts, usually impossible. I have, however, seen marked benefit obtained by the persistent use of viburnum prunifolium. This drug without doubt lessens the irritability of the uterine tissues, and thus diminishes the irritability of the source of irritation of the asthma.

The second group of drugs includes those that act on the nerve-centres, and thus inhibit reflex action. The most important of them are chloral, chloroform, ether, opiates, and bromides. When dyspnœa is intense a few whiffs of chloroform will give relief promptly. As the relief is often not of long duration, and as the drug cannot with safety be left in the hands of the sufferer, its range of usefulness is limited. Of this group, chloral is the safest and most universally useful. If the asthma is wholly paroxysmal, it is best administered in one or two full doses rather than in several smaller ones. Often 1.0 to 1.5 grammes (15 to 20 grains), given in sweetened water, will not only relieve present dyspnœa, but produce an effect sufficiently lasting to suppress the attack. In cases that complicate bronchitis, trachitis, or laryngitis, and in which the dyspnœa is not paroxysmal only, but to some extent is persistent, since the source of irritation is constantly excited, the best effects are obtained by the repetition of smaller doses of chloral, bromides, opiates, or of mixtures of all these with expectorants. In this way the nerve-centres are constantly inhibited or restrained in their activity, so that the paroxysms of exacerbation are held in abeyance, and time is gained in which to overcome the primary inflammation. A formula that I have frequently employed, with marked benefit in such cases, is the following:—

℞	Chloral., ...	grms.	15.00 (ʒiv).
	Ammonii muriatis, . . .	"	10.00 (ʒiiss).
	Morphiæ muriatis, . . .	grm.	0.20 (gr. iij).
	Antim. et pot. tart., . . .	"	0.15 (gr. iiss).
	Ex. grindeliæ robustatæ fl.,	c.cm. 45.00 vel	60.00 (℥iss).
	Aq. vel syr. glycyrrhiz.,	q. s. ad c.cm.	120.00 (℥iv).

Sig.: Give one teaspoonful every three to six hours, in sweetened water.

Morphia and the bromides are less generally useful than chloral. The bromides, given steadily, in rather large doses, are serviceable when the source of irritation is the larynx or pharynx, for they not only act favorably by lessening the excitability of reflex centres, but also have the peculiar property of benumbing the nerve-endings in the mucous membrane of the larynx and pharynx. The dose should be large; for example, 1.5 to 3.0 grammes (20 to 45 grains) of the bromide of sodium.

There is another class of remedial agents which it is difficult to classify with certainty, for our knowledge of their physiological action is imperfect, and the results of researches are not completely harmonious. The drugs to which I refer are grindelia robusta, senecio aureus, quebracho, lobelia, and tobacco. It seems probable, however, that as remedies for asthma they can be placed in this second group. Death from tobacco poisoning is due to paralysis of respiration. The physiological action of lobelia is very similar to that of tobacco. When lobelia is used in asthma it must be given in doses of from 2 to 4 cubic centimetres ($\frac{1}{2}$ to 1 drachm) of the tincture, and repeated every two hours, or oftener, until vomiting and relief are produced. Mitigation of the dyspnœa usually corresponds with intense nausea, and is greatest after vomiting.

Quebracho has a peculiar effect upon respiration in healthy persons. It slows it and prevents panting when hurried movements are made. At the same time it retards the heart. Gutman has shown that its active principle, aspidospermine, produces death by poisoning the respiratory centre.

We know less of the physiological action of grindelia and nothing of senecio. Grindelia produces death only in very large doses, and then by paralyzing respiration. In smaller doses it slows the respiration and the heart.

The effects of lobelia must be carefully watched, for large doses have produced alarming symptoms. For this reason I have employed it rarely, but grindelia I have administered frequently, and quebracho and senecio less frequently, although enough to feel confident that to some extent they are useful. They are so much less efficacious than some other remedies at our command for the relief of the dyspnœa that I rely upon them not at all for its treatment, but rather as adjuvants for warding off the recurrence of the paroxysms.

Grindelia and quebracho are probably mildly expectorant, and, through their bitterness, tonic to the stomach. But their bitter and otherwise unpleasant taste limits greatly their eligibility. Their fluid extracts can be administered in doses of 2 to 4 cubic centimetres (½ to 1 drachm).

The third group of drugs embraces the nitrites and nitro-glycerin. Amyl nitrite administered by inhalation has been used in asthma for a number of years. Nitro-glycerin has been used less frequently, and the nitrites of soda and potash still less. Prof. Fraser, of Edinburgh, has given us the most trustworthy information as to the relative value of these drugs in asthma. They all relieve the spasm, and with wonderful promptness. The effects of amyl nitrite are very transitory. Nitro-glycerin, when given in doses of sufficient size, is apt to provoke congestive headache. The nitrite of soda he found gave quite as prompt relief as the others, was less likely to provoke headache, and produced more enduring effects. The more purely spasmodic the case, the more efficacious are these drugs. Dr. Fraser found that, in two or three minutes after the administration of even half-grain doses of the nitrite of soda, marked relief was noticeable in the patient's breathing and a lessening of the crowing and piping in the chest. In ten minutes or less, patients feel comfortable. It was rare that it was necessary to repeat the dose in any single attack. The good effect of these drugs, when administered in the usual therapeutic doses, is undoubtedly chiefly due to their action upon the muscle-fibres of the bronchial tubes, irritability of which they lessen or temporarily destroy. In other words, they act upon the focus of irritation. In less degree they may diminish the irritability of the motor nerves. This is a somewhat doubtful effect of therapeutic doses, although it can be obtained from large doses. In using the nitrite of soda, which, from considerable personal experience, I can commend, it must be remembered that there are two preparations in the market,—a "commercial" and a chemically pure. The former can be given in doses of from 0.3 to 0.6 gramme (5 to 10 grains), and 1.3 grammes (20 grains) have been given without harm.* The therapeutic dose of the chemically-pure drug is from 0.06 to 0.3 gramme (1 to 5 grains). While the most beneficial effects are obtained in the most frankly paroxysmal cases, marked benefit may be derived from the continued use of these remedies in asthma that complicates bronchitis and that is to some extent persistent. In such cases I have combined the nitrite of soda with the usual expectorant and anodyne treatment of bronchitis. It may be administered in 18- to 30-centigramme doses (3 to 5 grains) every three to six hours.

Atropia, stramonium, and hyoscyamus constitute another series of drugs that are analgesic to the focus of irritation. The two first are the ones most frequently used in this disease. They cause relaxation of the bronchioles, in part by benumbing their involuntary muscular fibres and in part by lessening the sensibility of their terminal nerve-fibres. While efficient in aiding to give relief, their side-effects are so marked and often so disagreeable that they cannot be used in efficient doses. The action of the drugs is so well known that I need hardly say that these side-effects are: dryness of the mouth and throat, heat and redness of the skin, dilatation of the pupil, disturbed vision, and, in very susceptible patients, mental perturbations. Full doses are rarely employed, but frequently small doses are used to re-inforce the action of other drugs, as in the following formula, which is especially efficient for the relief of asthmatic dyspnœa:—

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* Therapeutics: its Principles and Practice. By H. C. Wood.

℞ Chloral., ...		grms. 20 (ʒv).
Sodii nitritis, . . .		grms. 3 (ʒj).
Tinct. stramonii, . .		c.cm. 6 (ʒiss).
Elix. simpl., .	q. s. ad	c.cm. 60 (℥ij).

Sig.: Take one teaspoonful every four hours, in water.

Not only do these remedies act favorably when they are taken by the stomach, but the inhalation of the smoke of the crude drugs is often of the greatest benefit. Stramonium-leaves are used in this way most frequently. The leaves are smoked either when rolled into cigarettes or from a pipe. Their efficacy is enhanced by first soaking them in a saturated solution of nitre and subsequently drying them for use; or they may be mixed with or rolled in bibulous paper that has been thus saturated. The nitre is decomposed by the heat, and a nitrite is formed which aids in relieving the dyspnœa. There are numerous proprietary cigarettes and pastels for asthma, the basis of whose composition is stramonium and nitre.

When uræmia is the cause of asthma, diaphoretics, diuretics, and cathartics are indicated for the elimination of the poison. The first of these classes of drugs gives the most prompt relief. Of diaphoretics, pilocarpine administered subcutaneously, is the most prompt in its action. It is necessary to prevent the re-accumulation of poisons thus eliminated. This is best accomplished by diuretics and, when they are not sufficient, the coincident use of cathartics. Cathartics and the preparations of jaborandi must be employed with caution when a patient is debilitated or has heart-weakness.

CHAPTER II.

TRACHITIS AND BRONCHITIS.

THE words trachitis and bronchitis are applied to acute and chronic catarrhal inflammations of the trachea and the bronchial tubes. Besides the simple form there are specific forms of bronchitis, such as tubercular. They are best considered separately. Usually, both the trachea and the bronchi are simultaneously affected, but in differing degrees of severity. The inflammation may centre in the trachea and scarcely involve the bronchi; the disease will then be recognized as trachitis On the other hand, if it centres in the larger bronchi, it is described as simple bronchitis, and, if in the smallest, as capillary bronchitis.

Anatomy of Acute Simple Bronchitis.—When the bronchial tubes are acutely inflamed, the first change that occurs is congestion of the submucosa, which is almost immediately followed by swelling, in part due to the congestion and in part to a serous exudate which fills the tissue. Simultaneously, the basement membrane thickens and the epithelial cells resting upon it are loosened from one another. If the irritation which excites the inflammation is severe, the submucosa fills with wandering round-cells of leucocytal origin. These cells, at first, are most numerous about the vessels, but soon fill the tissue diffusely, and in some cases penetrate and fill the muscular coat, and even the peribronchial connective tissue. They also mingle with the epithelial cells upon the surface of the bronchial tubes. Before this has occurred, however, the ciliated epithelial cells are cast off. The denuded surface is composed of embryonic epithelial cells, which, so long as the cause of inflammation exists and the process is active, are unable to reform ciliated columnar epithelium. So soon as active tissue destruction ceases, the epithelial surface is rapidly restored to a normal condition. The exudates, serous and cellular, are removed through the lymphatic channels, and perfect restoration is accomplished. While denudation of the epithelium is the rule in acute bronchitis, true ulceration is rare. The cells of the mucous glands are usually swollen, and the calibre of their ducts is diminished in part by this swelling and in part by compression by the swollen surrounding tissues. The occurrence and the degree of all these changes vary with the severity and duration of the inflammation. Many of them are wanting in the mildest cases.

The inflamed tissue is redder than natural, although the congestion is generally not evident to the naked eye when post-mortem examinations are made, and, if present, usually is seen in patches, and not uniformly distributed. The mucous surface of the trachea or the large bronchi, when inflamed, appears swollen and soft and has a dull gloss. In the opening stage of congestion it is unusually dry; later, it is covered with a larger amount of serum and mucus than is natural. At first the exudate is viscid and tenacious; soon, however, it becomes more dilute from the admixture of serum which partly forms it. It loses its early vitreous appearance, and is made whitish by degenerated and cast off cells. If the inflammatory process is severe or at all prolonged, the exudate becomes yellowish from the addition to it of round-cells of leucocytal origin, that resemble in all respects pus cells. The exterior of lungs in which the bronchi have been inflamed presents no abnormal appearances. When a section of them is made there flows from the cut surface, and chiefly from the ends of the divided bronchi, a frothy mucus. Muco-purulent matter, also, can usually be expressed from the latter. In simple acute bronchitis these changes are found to be symmetrically distributed in both lungs.

Physiological Disturbance.—The swelling of the mucous membrane of the trachea or the larger bronchi is not sufficient to cause noticeable obstruction to respiration. The unusually dry condition of the mucous membrane in the earliest stage of inflammation prevents the straining of the air, which is normally accomplished by the adherence of foreign particles to the mucus which covers its surface, and their removal by the cilia of the columnar epithelium. After desquamation has occurred, even though the surface is abundantly covered with mucus, dusts are imperfectly removed and may, therefore, prove the source of additional irritation.

Anatomical and Physiological Changes of Capillary Bronchitis.—In acute inflammation of the capillary bronchi the same anatomical changes are observable; but the difference in their normal structure leads to greater physiological perversions than are present when only the larger tubes are involved. The calibre of the smaller bronchi is proportionately very much less than of the larger, because of the deep infolding of the mucous membrane. For the same cause a transverse section of a bronchiole is stellate. Owing to these peculiarities the same degree of swelling which in the larger bronchi will cause no respiratory obstruction will greatly interfere with it in the smaller ones; and a moderate amount of mucus, cast-off epithelium, and detritus from cellular degeneration will produce complete obstruction. Dyspnœa, more or less intense, is, therefore, a characteristic of capillary bronchitis. There is a decided tendency, also, and especially in very severe and in chronic cases, for the inflammation to extend through the bronchiole to the surrounding lung-tissue, and to produce peribronchitis or catarrhal pneumonia.

Complete obstruction of the bronchioles leads to a circumscribed collapse of the alveoli communicating with them. (See page 67.) It is rare that such patches of collapsed lung are large, or even very numerous. Partial obstruction leads to the production of emphysema. It is difficult for air to pass through the inflamed bronchioles, both in expiration and inspiration; but as the former is almost wholly a passive act, being produced by the elasticity of the lung and by the weight of the thoracic and abdominal walls, it is not a very forcible one; but the latter is an active one produced by muscles of considerable power, and, accordingly, is much the more forcible. Therefore, air will enter the lungs through partly obstructed bronchioles, but will be very imperfectly emptied through them. Thus, slowly an increment is constantly added to the alveolar contents, and dilatation ensues. (See page 61.)

Emphysema is exceedingly common as a complication of bronchiolitis; indeed, it is to some extent a constant accompaniment of it. It may involve the lung quite diffusely. The production of emphysema in bronchiolitis is aided by the increased air-pressure which is produced in the tubes by severe coughing.

A frequent, severe, and dreaded complication is catarrhal pneumonia. (See page 82.) This is developed in part by an extension of inflammation from the bronchioles to the alveoli communicating with them, and in part by an extension through their walls to the contiguous alveoli. Minute and infrequent patches of consolidation thus produced are often encountered in cases of capillary bronchitis. Occasionally they are very numerous, and form a severe complication. They rarely coalesce in sufficient numbers to produce extensive areas of consolidation.

Anatomy of Chronic Bronchitis.—The anatomical changes wrought by chronic bronchitis are various, and occur, in the same case, in varying combinations and in different parts of the bronchial system. The mucous membrane is usually grayish or brownish red. It may be thickened. Papilliform outgrowths and excrescences are not uncommon. Unusual thinness of the mucous membrane is quite as characteristic as increased thickness. Especially in sacculated dilatations the epithelium is apt to be pavement-like and the surface to resemble more a serous than a mucous membrane. The connective tissue is uniformly hypertrophied to a greater or less extent. When acute exacerbations of the chronic trouble occur, the submucosa is filled with round-cells, and all the usual changes incident to acute inflammation are observable in the other bronchial tissues. Peribronchial inflammation is usually present in severe cases, especially if the smaller bronchi are involved. As a result of it the bronchial wall seems greatly thickened and supported at points by considerable masses of firm connective tissue that extend out into the neighboring lung-structure. This connective tissue develops from a thickening of the alveolar walls by inflammatory exudates. In its formation many alveoli are obliterated; others are narrowed and distorted. The muscular layers of the bronchi are often atrophied and the individual fibres separated by new connective tissue. In the larger tubes the cartilages are also at times atrophied. The bundles of elastic fibres, which are more or less numerous in the bronchi, remain unchanged or are hypertrophied. Occasionally the interior of a bronchus will present a fenestrated appearance, the mucous surface being ridged both longitudinally and transversely. This appearance is due to the atrophy of the bronchial wall and especially of the muscular fibres, and to the unchanged condition or hypertrophy of the elastic fibres which produce the ridges.

The mucous glands may be quite normal in appearance. Often, however, they are changed. Many atrophy. They may even disappear entirely. Sometimes the duct becomes funnel-shaped and opens with a wide mouth upon the surface of the bronchial tube. In other cases it is obstructed or compressed by the swollen surrounding tissue, and consequently the deeper portion of the gland is converted into a small retention-cyst.

Abrasions of the mucous surface in chronic bronchitis are numerous and often extensive. Ulceration is not rare, but usually is limited to dilated portions of the bronchi.

Moderate chronic emphysema is the rule. When the larger bronchi alone are involved this lesion is chiefly confined to the anterior and lower border of the lungs. It is then due to severe, frequent, and prolonged coughing. When the smaller bronchi are involved the emphysema is more extensive and more generalized, and is due to the same causes that produce it in acute bronchiolitis.

A frequent complication is bronchiectasis. (See page 57.) The dilated bronchus is usually cylindrical in shape, but may be sacculated and very large. Ulceration upon the surface of the dilatation is of frequent occurrence. It may lead to an enlargement of the cavity, and rarely permits its contents to escape into the lung-tissue by penetrating the bronchial wall. This accident almost invariably provokes gangrene of the lung. (See page 112.)

The contents of chronically inflamed bronchi vary greatly. They may contain tenacious, adhesive, vitreous mucus in slightly larger quantities than is normal. More frequently they contain a larger amount of frothy mucus, which is not very adhesive, and in places some mucopurulent matter. Rarely, a bronchial dilatation will contain enormous quantities of serous fluid or pus. Either of these exudates may become fetid from the occurrence of putrefaction within the bronchi.

Symptoms of Acute Trachitis and Bronchitis.—All grades of severity of inflammation of the trachea and the larger bronchi are observed, from a cold so mild as to be almost unnoticed to one that causes fever and general disturbances that necessitate confinement to the house and often to the bed. In the severer cases, both of trachitis and bronchitis, the sufferer observes the same subjective symptoms. They are distinguishable only by physical signs.

In moderately severe cases of these affections their onset is usually marked by a sensation of chilliness, or sensitiveness to draughts or changes of temperature, and more rarely by an actual rigor. The transitory but constantly-recurring chilly sensations are felt for several hours. During this time the patient often sneezes and begins to feel a mental and physical heaviness or languor. Actual headache of a dull character is frequently felt. Cough is an early symptom, and at first it is not severe, but rapidly increases in severity. With it, and even with deep breathing, a feeling of rawness and soreness is felt beneath the sternum, which is due to the tracheal inflammation. The nose and throat may or may not be simultaneously inflamed.

As the chilly sensations disappear fever develops, and the skin becomes dry and hot. The temperature is rarely high except in children. Coincidently and dependent upon the increase of temperature, the pulse becomes quick and respiration slightly hurried. A general muscular aching over the body is felt. On the second day the soreness beneath the stĕrnum is more constant. The cough is more severe and frequent. Often it is sufficient to cause muscular soreness about the waist, in the epigastrium and loins. As yet the cough is unaccompanied by expectoration. On the third day, or often a little earlier, a scant, vitreous, slightly frothy sputa is expelled by coughing. It rapidly becomes more abundant and more easily raised. Usually, it is more frothy and in part muco-purulent. The fever now disappears. The tracheal soreness is much lessened or gone; and as the cough, though still frequent, is less severe, the abdominal muscular soreness disappears. Recovery is usually completed by the seventh or tenth day.

So long as the fever lasts, appetite is wanting or capricious. The bowels are usually inactive. The urine is somewhat diminished in quantity and dark in color.

A physical examination demonstrates the involvement of the bronchi; but in simple trachitis no abnormal physical signs are discoverable in the thorax. If the chest is examined in the latter cases the only abnormality observable may be a quickening of the respiratory movements when the patient is feverish. When there is bronchitis, vocal fremitus is usually normal. Rarely, a bronchial fremitus can be felt. Resonance is natural. Auscultation reveals in the early stage dry râles, which are coarse when the large tubes only are inflamed, and fine when the smaller ones are. Later, when the exudate within them becomes thinner, moist râles, coarse or fine, according to their location, are audible. The moist râles are not heard continuously in one place, but may disappear after coughing, to be discovered elsewhere; or, they may be heard for a few moments, disappear, and not re-appear for many minutes. If the exudate is very abundant and the inflammation quite diffuse, the râles may be continuous.

Symptoms of Acute Capillary Bronchitis.—Acute capillary bronchitis oftener begins with a rigor of some duration, which is followed at once by fever. Frequent, dry, and hard coughing is at once noticeable. The breathing gradually becomes quicker, more labored, and productive of the subjective sensations of dyspnœa. In thirty-six hours, and often much earlier, the disease has become severe, and endangers life. The patient's countenance is anxious and disturbed. The face is somewhat congested; the lips full, and, as the dyspnœa deepens, livid; the nares open widely with each inspiration. The rapidity and labor of breathing increases. It is often noisy, expiration producing a prolonged wheeze or whistle. So difficult is breathing that a sitting posture is constantly kept rather than a reclining one. Young children are most comfortable when held or carried. The cough continues frequent, harsh, and wearying. It is accompanied by little expectorate, and in young children usually by none. The skin is hot and dry; the temperature ranges from 101° F. to 103° F., the higher temperature being most frequently observed in children. The temperature is highest at night, but is usually nearly or quite normal in the morning. The pulse is quick; at first full and firm.

If the disease tend to a fatal termination the dyspnœa is greater and cyanosis is marked. The face is ashen; the lips and finger-nails are purplish; the skin is cold and clammy. The pulse grows small, soft, and quicker. The patient is somnolent or nearly unconscious; is now too feeble to sit, and lies upon his back, with his head buried deeply in the pillow, and mouth open. Respiration grows shallow; less wheezing is heard, but a constant rattle in the throat replaces it. Respiration becomes irregular for a few minutes, a few convulsive movements pass over the body, and breathing ceases. In these, the commoner cases, death is caused by suffocation. Less frequently, heart-failure is its immediate cause.

Although capillary bronchitis must be regarded as one of the severe acute diseases, fortunately a large proportion of those who suffer from it recover. Improvement usually begins by the third or the fifth day, although at times it begins several days later. The cough is easier and more satisfactory, as with it an expectorate is raised. This at first consists of small lumps of vitreous mucus, but soon consists of large quantities of frothy mucus, in which are imbedded small muco-purulent chunks. If this latter expectorate is thrown on water the frothy mucus floats, and from it dangle minute muco-purulent strings, which have been formed in the small bronchi. The fever gradually lessens. Respiration is less hurried and difficult. The skin is no longer hot and dry, but natural. The appetite improves. Strength is gradually regained. Somewhat slowly health is restored.

In severe acute attacks the sufferer is much weakened and often considerably emaciated. In less severe cases and in those that are subacute or chronic, nutrition is little interfered with. Relapses are especially frequent in capillary bronchitis, and must be guarded against with unusual care.

Physical examinations show a chest that is enlarged. In severe cases the chest is constantly in the inspiratory position. During respiration the ribs move little; the intercostal spaces are persistently stretched, and inspiration is produced by a lifting of the entire chest by the unusual muscles of respiration. The degree to which these changes develop depends upon the severity of the attack and upon the amount of coincident emphysema. The frequency of respiration is increased in proportion to the intensity of the dyspnœa. In young children the lower ribs and the abdomen are deeply pressed inward during inspiration, the intra-thoracic pressure being low, because of the bronchial obstruction. In adults, in whom the ribs are firm and little pliable, the intercostal spaces only are pressed in, and the abdominal wall is crowded back toward the spine. Tenderness over the chest is often noticeable, and especially in children. No abnormal fremitus is observable unless a complication has caused somewhat extensive consolidation of lung-tissue. On percussion, resonance is found to be normal or, from the emphysema, somewhat increased, and especially increased in area as the dilated lungs crowd the liver and heart downward and overlap the latter. Auscultation reveals fine râles, either moist or dry. The vesicular sounds are obscured by them or are lacking. The inspiratory sound is short and the expiratory feeble and prolonged. These modifications of the respiratory sounds are best heard over the lower and posterior parts of the chest. The characteristic fine râles are often noticed, or are mixed with coarse ones, when the large bronchi are simultaneously affected.

When the common complication of catarrhal pneumonia (see page 82.) exists the physical signs are rarely modified; for the consolidation of lung-tissue does not involve areas of sufficient size to produce increased vocal fremitus, dullness, or bronchial respiration. Sometimes, however, the affected lobules are so close together that physical signs of consolidation can be discovered. Catarrhal pneumonia causes an increase in temperature if it is at all extensive, the evening temperature ranging then above 103.5° F.; and all the symptoms are intensified, though not changed in character. Many mild cases of capillary bronchitis are seen in which the symptoms that have been described are observable, but in less severe or in a modified form.

Symptoms of Chronic Bronchitis.—Inflammation of the bronchial tubes may be chronic in character from the first, or acute bronchitis may become chronic. There are also numerous subacute cases which present a mixture of the symptoms of those that are acute and chronic. They last several weeks. Chronic bronchitis is of very frequent occurrence. Many symptoms are common to all cases and many more are variable. It is therefore a disease whose physiognomy is multiform. Its duration is indefinite. Often it will last for ten, twenty, and even more years. It does not lead to fatal results except when complicated by other ailments. Its course is not one of uniform severity, but frequent exacerbations and remissions are the rule. Different varieties of the disease also vary in severity. In general, we may say that, the longer it lasts, the severer grow the symptoms which are constantly present. Often the exacerbations resemble in all respects acute bronchitis.

The state of general nutrition of those who suffer from chronic bronchitis varies greatly, and is not dependent upon the disease. We often find chronic inflammation of both the large and small bronchi in those who maintain, nevertheless, an unusual proportion of flesh and are in all ways robust. When, however, it occurs in aged people, it usually prevents the accumulation of fat, and exacerbations cause loss of both weight and strength, which is with difficulty, if at all, recovered from. Vomiting, which often occurs immediately after eating, because of hard coughing, is seldom sufficiently severe to interfere materially with the maintenance of strength. The putrid and purulent varieties of bronchitis are wasting diseases, and in this respect resemble phthisis.

Uncomplicated chronic bronchitis is an afebrile disease. Purulent and fetid bronchitis are usually accompanied by fever of a hectic type. The pulse is normal in rapidity and character unless sudden or violent exertion or fever has quickened it. Changes in the heart are not very common; rarely its right ventricular wall hypertrophies because extensive peribronchitis interferes with the pulmonary circulation. Pain about the chest is not usual. In the milder cases, when acute exacerbations occur, tracheal and, therefore, substernal soreness is felt during breathing and coughing. Although in chronic bronchitis the cough is more apt to be very severe and prolonged than in acute, muscular soreness is seldom complained of about the waist or line of attachment of the diaphragm. This is because the muscles have become accustomed to the strain, and have hypertrophied in consequence of it. The muscles which most frequently hypertrophy in chronic bronchitis are the recti abdominalis and the sterno-cleido-mastoids,— the first because of the persistence and severity of the cough, and the second because of dyspnœa, which, in some cases, is great. All who suffer from chronic bronchitis notice an unnatural shortness of breath on exertion. If the bronchioles are involved or if there is extensive peribronchitis, dyspnœa, usually slight, though varying much in severity, is felt constantly. Ordinarily the respiratory movements are not unnaturally quick, but are hurried if sudden and rapid movements are made and also if dyspnœa is considerable.

Coughing occurs more or less frequently each day. In the mild cases it is so infrequent and slight as to escape the sufferer's notice. When severe it is apt to be harsher, more prolonged, and more distressing than in acute bronchitis; but its character is very variable.

The character of the expectorate of those suffering from chronic bronchitis is perhaps the best index of the nature of the changes that are taking place within the bronchi. Therefore, upon its peculiarities are based the varieties which are usually described.

1. *Simple chronic bronchitis*, or, as its milder form is often called, *chronic winter cough*, is most common. When mild, there is so little coughing or respiratory disturbance during the summer that the existence of a bronchial affection is not suspected. But each fall the cough is aggravated, and is troublesomely persistent until the following summer. Such winter exacerbations and summer remissions may occur for years. Usually, as summer follows summer, the persistence of the cough throughout the year is more noticeable. Occasional acute exacerbations will occur in hot weather, and greater shortness of breath, when hurried movements are made, attract the sufferer's attention. In the severer cases coughing is frequent and hard at all seasons; dyspnœa is troublesome; sleep is usually disturbed by coughs; prolonged or severe physical exertion is impos-

sible; and occupations that necessitate exposure during inclement or changeable weather or the inhalation of irritating dusts must be abandoned. The amount of expectoration usually varies with the severity of the attack. It is muco-purulent or, in mild cases, frothy. It is most abundant and purulent in the morning. Usually, if purulent in the morning, it becomes frothy and vitreous during the day. Coughing is almost invariably harshest in the morning, on first arising, and at night, on first retiring. Frequently it is provoked by going into rooms or atmospheres of different temperatures.

2. *Dry bronchitis* occurs less frequently than the preceding variety. It is characterized by the absence of expectorate or by the occasional discharge of a small clump of tough, vitreous mucus, which is loosened and dislodged with difficulty. The cough is especially inclined to be harsh and paroxysmal. Asthma and chronic emphysema are frequent complications of it.

3. *Bronchorrhœa* constitutes a third variety. Bronchiectasis always co-exists with it. In the dilated tube there rapidly accumulates a large amount of serous fluid, which is expelled periodically by coughing. The periodicity of the cough is very marked in most cases, and occurs just so soon as a certain quantity of fluid accumulates. The cavity is often emptied more frequently and more perfectly if the sufferer lies upon the side in which there is no cavity. In rare cases the quantity of expectorate is enormous. It may be several pounds daily. Its amount, its serous character, and the periodicity of its expulsion are its important peculiarities.

4. *Purulent bronchitis* also occurs when bronchiectasis exists and when the wall of the cavity is exten-

sively ulcerated. The sputa is moderate or large in amount, and consists of thin pus. It is not raised in formed lumps, which float separately in a sputa-cup, but as mouthfuls of liquid pus, which at once coalesce in the cup, and resemble pus from an abscess.

5. *Putrid bronchitis* is characterized by putrefaction of the contents of the bronchi. This may be a complication of either of the other forms, but most frequently is associated with bronchorrhœa. It is most apt to develop when the bronchi are imperfectly emptied and the secretions stagnate in them. It leads, in serious cases, to sloughing of the bronchial walls and sometimes to gangrene of the lung. When severe it is a wasting disease, accompanied by hectic fever. It may terminate in a few weeks if gangrene of the lung is produced, or it may last for many months. Mild cases sometimes recover. The contents of the bronchi are so foul that the breath becomes excessively offensive. Its odor is often plainly detectable by those standing several feet from the sufferer. It usually causes lessened appetite and sometimes nausea and vomiting. The expectorate is considerable or very large in amount. If collected in a sputa-cup it separates, on standing, into three layers. The uppermost is frothy; the middle is a serous fluid; the lower consists of opaque, granular matter. At the bottom roundish, grayish-yellow balls are also found, varying in size from a pin-head to a pea. These are extremely offensive, especially if compressed. Under the microscope they are found to consist of granular matter, fat-crystals, micro-organisms of various kinds, and filamentous growths. These bodies are called mykotic plugs, and are supposed to be the cause of the putrefaction. The sputa ceases to emit odor after it has stood for a time, but does again if it is agitated.

The physical signs necessarily vary somewhat with the severity and form of the bronchitis. Increased rapidity of respiration is noticeable only after greater or less physical exertion, unless emphysema is considerable. If the bronchitis has long been chronic, thoracic expansion is always imperfect. The subjective symptoms of dyspnœa are not felt in uncomplicated bronchitis. No abnormal fremitus is felt by the palpating hand unless, from extensive peribronchitis, considerable consolidation of lung-tissue has occurred. Thoracic resonance is also normal if complications are absent. Auscultation reveals the most positive physical signs. In the milder cases the normal respiratory sounds are exaggerated and roughened. In these cases occasional moist râles may or may not be heard here and there. An expiratory sound is almost always present, Its length and intensity vary much. In the severer cases vesicular respiratory sounds are inaudible because moist or dry râles obscure them. So frequently are complications present that the sounds are often further modified by them. If the bronchioles are involved and emphysema is considerable, whistling and crowing sounds obscure all others. Bronchiectasis may produce the physical signs of a cavity. Peribronchitis may cause lung consolidation, which can be recognized. A peculiarity of the physical signs of uncomplicated chronic bronchitis is the uniformity of their distribution over all parts of the chest, or at least their symmetrical distribution on the two sides.

Diagnosis.—It is not difficult to recognize simple acute bronchitis. It is rarely mistaken for any other disease. At times a pharyngitis, laryngitis, or trachitis, which produces a severe cough and some systemic disturbance, may be mistaken for acute bronchitis.

The absence of physical signs of disease of the bronchi and evidence of disease of the upper portions of the respiratory tract should rectify the diagnosis. More rarely an acute exacerbation of a mild chronic bronchitis is mistaken for the simple acute inflammation. This only occurs when the history of the case is imperfectly obtained. Occasionally we must differentiate between acute bronchitis and phthisis; more frequently between chronic bronchitis and phthisis. We can, however, best discuss the distinguishing characteristics of each after describing phthisis. (See page 138.)

The characteristic symptoms of capillary bronchitis and asthma have been described (see page 9) and emphysema will be. (See page 61.)

Causes.—Bronchitis in all its forms is influenced by the same causative factors, although in varying degrees. It is both a primary and a secondary disease. When a secondary disease it sometimes results from mechanical interference with the circulation of the blood through the bronchial vessels, and sometimes from poisonous and infectious matter that is inhaled and causes the bronchitis and subsequently a disturbance of the whole system. Illustrations of the latter group of cases are seen in the bronchitis of measles and whooping-cough. Many cardiac lesions cause persistent, passive engorgement of the lungs and bronchi, and lead to an inflammation of them.

Cases of primary bronchitis are the result usually of both predisposing and exciting causes. It occurs at all ages. It is sometimes said to occur oftenest in childhood and old age. My own experience does not confirm this statement. I believe it occurs with about equal frequency at all ages. It is a more dangerous disease in infancy and old age than at other times.

Debility, which lessens the power of man to resist disease, is one of the most frequent predisposing causes. We therefore find bronchitis of common occurrence during convalescence from other troubles. Anæmia, rheumatism, Bright's diseases, and diabetes seem especially to make those suffering from them susceptible to bronchitis. Bronchitis itself, more than any other disease, makes one susceptible to repeated attacks.

Enervating habits are frequently the cause of increased susceptibility to the disease. Sedentary habits, and especially if they necessitate the constant breathing of dry, warm air, predispose to all forms of simple inflammation of the respiratory passages; for the change which those subjected to such surroundings must experience on passing from the dry, warm, and often close air of the house or office to the moist, cold air outside, such as exists in temperate climates during the fall, winter, and spring, is greater than any atmospheric change ordinarily produced by nature. Air laden with impurities is always irritating to the respiratory passages; therefore, a life of confinement, in poorly ventilated rooms, increases one's susceptibility to bronchial inflammation.

The excessive use of alcohol, and even its steady moderate use, is a prolific source of inflammation of the respiratory passages. Continuous excess causes enervation and decidedly lessens the power of the human organism to resist disease of all kinds. The benumbing effect of alcohol causes an insensibility of those using it, to cold and atmospheric changes, and to other exciting causes of bronchitis, so that they protect themselves imperfectly.

There are occupations which predispose to bronchitis. They are such as necessitate the inhalation of dust or certain gases. The coal-miner, the stone-cutter, and the grain-shifter are each exposed to dusts that are frequently exciting causes of the disease. Many persons who are employed in foundries, in boiler-rooms, and gas-houses are subjected to great heat at times, and through carelessness expose themselves to rapid cooling. Among such persons bronchitis or affections etiologically allied are of constant occurrence.

The exciting causes of bronchitis are (1) most frequently atmospheric changes, and (2) less often irritating dusts or gases. The atmospheric conditions which most frequently provoke it are sudden depressions of temperature in a moist atmosphere. Bronchitis occurs relatively much oftener in a moist than in a dry atmosphere, for the withdrawal of heat from the body takes place much more rapidly when the air is filled with moisture and a sudden chilling of the surface may be thus produced. In a moist air a sudden fall of temperature of only a few degrees will be felt more keenly by the human system than a fall of many degrees in a dry air.

These atmospheric conditions occur oftenest in the spring and fall, and therefore we find cases of acute bronchitis or acute exacerbations of chronic bronchitis most frequent in these seasons. They are least frequent in the dry cold or hot portion of winter and summer. They are also less frequent in years in which the summers are cool and damp, and in which there are not great extremes of temperature or other atmospheric conditions.

Bronchitis is more or less prevalent in all climates, but least so in the tropics and most so in temperate regions, if we except from the latter such localities as are characterized by great dryness.

Dusts and gases, which are exciting causes of bronchitis, vary in their degrees of irritating power with their character. For example, dusts of vegetable origin are the most irritating, those of animal origin a little less irritating, and mineral dusts least so. Very few cases result from the inhalation of gases, and the most irritating gases, such as bromine, chlorine, etc., are not commonly met with.

Capillary bronchitis is produced by the causes already enumerated. Often it is the result of the extension of inflammation from the large bronchi to the small ones. The exanthemata and whooping-cough among acute diseases, and Bright's disease and weak heart among chronic ones, especially predispose to it.

Chronic bronchitis is peculiarly apt to occur in those who have rheumatic, gouty, and scrofulous diatheses or diabetes.

Treatment.— The *prophylactic treatment* which is applicable to bronchitis is almost equally applicable to all its forms. Many attacks of acute bronchitis could be prevented by correcting enervating habits and diseased conditions which predispose to bronchial inflammations. Not only can acute attacks be often prevented, but the tendency for acute inflammation to become chronic can in the same way be counteracted.

The deleterious effects of atmospheric changes that are exciting causes of the disease can be avoided by keeping the skin covered with woolen underwear, which maintains it at a comparatively equable temperature. Dwelling in rooms whose air is artificially highly heated and dried should be avoided at all seasons. The best indoor winter temperature for those who are vigorous is 68° F., and it should not be permitted to exceed 70° F. The air of offices and dwellings should be kept fresh by careful ventilation, for stale air is both enervating and often directly irritating to the air-passages of man. Especially should these directions be kept in mind by those who are prone to bronchitis.

The breathing of dust-laden and irritating air must be avoided by all who are predisposed to the disease, and especially by those who suffer from chronic bronchitis. This often necessitates a change of occupation. Grain-shifters and others who must breathe irritating dusts can frequently avoid its effects by wearing a respirator. The simplest is a sponge fastened beneath the nostrils and over the mouth, through which the inspired air is strained.

Medicinal treatment must be varied with the form of the bronchitis and with the stage of the inflammation. To lessen the frequency and severity of coughing, to modify the secretions and excretions formed upon and within the inflamed tissues, are almost universal indications for treatment in bronchitis. To meet the first of these, opium or its anodyne alkaloids are chiefly relied upon. The bromides are often used instead for children, but they are not as efficacious. Chloral, administered alone or coincidently with opiates, is exceedingly useful when coughing occurs in paroxysms of nervous origin. If, however, the paroxysms are caused by an accumulation of secretions in dilated bronchi, chloral proves no more efficacious than other anodynes.

A large number of remedies may be used to modify the secretions and excretions. Ammonium chloride and carbonate are administered, in order to render mucous secretions thinner, less adhesive, and more easily dislodged. The carbonate is preferable when the finest bronchi or lung-tissue is involved, but there is little choice when the larger bronchi are inflamed. The nauseating expectorants, such as tartar emetic, preparations of squills, and others, act partly as do the ammonia compounds, but are most useful in aiding the expulsion of secretions from the bronchi. It is seldom necessary to resort to vomiting to empty the respiratory passages of secretions.

In chronic bronchitis it is often desirable to stop suppurative inflammation. The purulent character of sputa can be promptly and to a most marked extent diminished by the internal administration of turpentine, Venice turpentine, creasote, and similar drugs. The fetid quality of bronchial secretions is best counteracted and its persistence prevented by the conjoint administration by the mouth of creasote and terebinthines and by the inhalation of volatile antiseptics.

In the chronic forms of bronchial inflammation the prevention of the development of new tissue and the thickening of the bronchial walls by the round-celled exudate which occurs is a common indication for treatment. The iodides exert the greatest influence over these conditions. Their steady employment apparently hinders the development of adult from embryonic tissue. The iodides of sodium, potassium, and ammonium are also mild expectorants, acting as do the chloride and carbonate of ammonium. Of the iodides the ammonium compound is the best expectorant, but is most irritating to the stomach. Therefore, as they must in many cases of chronic bronchitis be employed continuously for long periods, the iodide of soda is usually to be preferred.

Undoubtedly, many threatened attacks of bronchitis can be checked or rendered abortive. But this is only possible in the stage of congestion, before inflammation is established, and providing the exciting cause is removed. If, just as the chill is passing off and tracheal soreness and coughing begin, diaphoresis is promptly produced and simultaneously a full dose of opiate is administered, a cessation of the development of the disease can be obtained. Dover's powder is the opiate that is best adapted to this stage, and should be given in a single large dose. Diaphoresis should be established at the same time by hot baths and drinks. The opiate undoubtedly lessens the irritability of the congested tissue, and the diaphoretics deplete the congested vessels and prevent the formation of exudates and other phenomena of inflammation. This mode of treatment is well adapted to sthenic cases only. If the patient is debilitated from any cause, if his tissue-changes are taking place slowly, a different abortive treatment proves more effectual. This consists in the administration of one or two large doses of quinine and an opiate. In these cases there is a lack of vascular tone, and, therefore, depletion of the congested vessels does not lead to their contraction and restoration. Quinine, however, and strychnine also, seem to stimulate the vasomotors, so that the lost vascular tone is regained. The opiate is still needed to lessen the irritability of the bronchial tissues. A capsule of quinia sulphate and morphia sulphate, in doses of 0.3 gramme and 0.008 gramme (5 and $\frac{1}{8}$ of a grain), respectively, is the most convenient form of administration. Two of these should be given at first, and one every three or four hours afterward, until four or five doses have been taken.

Unfortunately, abortive treatment can rarely be applied, as patients do not often seek a physician until the inflammation is fairly established and until it is too late to employ it successfully.

In the dry stage of *simple acute bronchitis* the indications are (1) to lessen cough and soreness, (2) to promote the formation of a thin expectorate, and in the moist stage (3) to aid the elimination of secretions. As the course of the disease is usually short, a formula is generally advised which will meet all of these indications at once; such as the following:—

1. ℞	Ammon. muriat.,	grms.	10.0 (ʒiiss).
	Morph. muriat.,	grm.	0.2 (gr. iv).
	Antim. et pot. tart.,	grm.	0.15 (gr. iiss).
	Syr. glycyrrhiz., q. s. ad	c.cm.	120.0 (℥iv).

Sig.: Give of the mixture one teaspoonful to adults every three to six hours.

2. ℞	Sod. bromid.,	grms.	20.0 (ʒv).
	Tinct. sanguinariæ,	c.cm.	10.0 (ʒiiss).
	Tinct. opii camph.,	c.cm.	45.0 (℥iss).
	Syr. scillæ comp.,	c.cm.	45.0 (℥iss).
	Syr. tolut., q. s. ad	c.cm.	120.0 (℥iv).

This can be given in the same doses as the last, but more frequently, if needed. All expectorant mixtures are more or less nauseating, and diminish the appetite and make digestion slow. The second formula, while less efficient as a cough-mixture, causes very little disturbance of the stomach. I have found frequently that when the stomach is peculiarly sensitive the following capsule proves efficient:—

℞	Ammonii chloridi,	grm.	0.06 (gr. j).
	Codeiæ,	grm.	0.012 (gr. $\frac{1}{5}$).

One should be administered each hour. When the larynx and trachea are chiefly involved, a compressed tablet of the following ingredients is exceedingly useful:—

℞	Terpin. hydrat.,	grm.	0.12 (gr. ij).
	Extract. cannabis Indicæ,	grm.	0.003 (gr. $\frac{1}{20}$).
	Codeiæ,	grm.	0.008 (gr. $\frac{1}{8}$).

They may be given alone, one every half-hour or hour, with less frequent doses of expectorant mixtures. They should be allowed to melt gradually in the mouth.

The fever of bronchitis rarely requires especial treatment. In adynamic cases, or when debility is a prominent feature, moderate doses of quinine are useful (from grm. 0.15 to 0.25). In sthenic cases aconite may be used, and may contribute materially to lessen fever and to produce a feeling of greater well-being. To children who sometimes have high temperature and consequent delirium, a few doses of antipyrin can be given most advantageously.

Many mild cases of acute bronchitis require no treatment, and numerous complications necessitate a modification of the course just sketched. It is desirable that the bowels be kept regular. As the opiates which are usually required cause constipation, a laxative must be administered.

The indications for treatment in *acute capillary bronchitis* are (1) to maintain strength, both (*a*) general and (*b*) cardiac; (2) to relieve dyspnœa by (*a*) loosening and (*b*) expelling the secretions from the bronchioles.

The course of simple acute bronchitis is so short, and ordinarily the general strength of a patient is so little affected, that special dietary directions are unnecessary. In capillary bronchitis, however, very great and rapid physical prostration is produced. Often, cardiac weakness is developed, which greatly increases the danger to life. For these reasons the sufferer's strength must be preserved. A diminished appetite, and even a disinclination for food, is the rule in this disease. Therefore, feeding should take place with as much regularity as the administration of medicine, and should not be governed by appetite. The food should be concentrated, and in such a form that it is most easily digested. Milk and milk-gruels are probably the best foods. Occasionally, to vary the diet, meat-broths can be advantageously given, or soft cooked eggs or raw eggs beaten up in the milk. The food is best administered in small quantities during the severest part of the attack, and frequently repeated, so that the stomach, which is doing its work slowly, may not be overloaded. And even when disinclined for food a patient will take two or three teaspoonfuls without disgust. Especial attention must, in severe cases, be paid to the maintenance of strength.

Cardiac strength must be preserved, whenever acute failure is threatening, by the combined use of diffusible stimulants and cardiac tonics. The carbonate of ammonia is one of the best diffusible stimulants. Its effects are the most promptly obtained when it is given in solution, and they may be somewhat augmented by dissolving it in camphor-water. The doses should be such that they can be repeated as often as every hour, for the stimulant effect of the drugs is transitory. Excellent results can be gotten from a solution of camphor in oil administered hypodermatically. The effects of alcoholics under these circumstances can be best considered in connection with the treatment of croupous pneumonia. (See page 103.) If used, alcohol should be given diluted, so that its exact strength is known. Its stimulant effects on the circulation are transitory and are obtained only from small doses. So soon as its anæsthetic effects are developed, either from the administration of large doses or small ones, frequently repeated for some time, cardiac stimulation is no longer obtained, and the patient is less likely to keep the air-passages clear by coughing and voluntary efforts to breathe deeply; congestion is prolonged and tendencies to congestion confirmed because of the lessened vascular tonicity which is produced. Furthermore, alcohol in the blood lessens its ability to take up oxygen from the air inspired. In a word, it increases the difficulty already existing of furnishing to the tissues the oxygen that they need. Digitalis, or strophanthus, which is often better in these cases, must be used simultaneously with the diffusible stimulants. As a rule, before the latter drugs are indicated, when the pulse is rapid and begins to grow soft or small, digitalis, or one of its congeners, should be employed to stimulate the heart's contractions and prevent the development of marked cardiac weakness. Veratrum and aconite should never be used unless at the very onset of an attack, and then they may act as depletors might,—to relieve congestion and prevent inflammation. If used while the heart is laboring and growing weary, they increase the tendency to cardiac dilatation. They are hardly ever required in bronchiolitis.

To relieve dyspnœa, the air-tubes must be emptied of the obstructing secretion and the swelling of the walls of the finer ones lessened. The first of these indications for treatment can be met by rendering the secretions more liquid, and therefore more easily dislodged. The carbonate and muriate of ammonia will help to accomplish this. The inhalation of moist air is exceedingly beneficial, as all the moisture inhaled at once aids by diluting the secretions. Those suffering from severe attacks of bronchiolitis should be kept in rooms whose air is at a constant temperature and made moist by evaporating water. The inhalation of the warm spray of a steam-atomizer is also useful. The patient can

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render the secretions less tenacious, but also more copious, by free libations of water and fluid foods.

Emesis is seldom advantageous. By it the bronchial tubes are most perfectly emptied, but the depression which most emetics produce is deleterious, and often is followed by an irritable condition of the stomach which interferes with the maintenance of nutrition. The subsulphate of mercury causes prompt emesis with very little depression. It can be used when there is much dyspnœa in strong and vigorous patients, but it should not be frequently repeated. Oftener emetics are given in small doses as nauseating expectorants. Apomorphia is a favorite with many, and is given in doses of 0.003 gramme, or one-twentieth of a grain, every two to four hours. Similarly tartar emetic (dose, grm. 0.005, or gr. $\frac{1}{12}$) and syrup of squills and ipecac (dose, c.cm. 2 to 4, or ʒss to j) can be employed.

Forceful respiration is one of the principal means by which mucus is dislodged from the bronchi. In dyspnœa, when voluntary exertion flags and when cyanosis develops, deep and forceful breathing is stimulated by sudden immersion in a bath or by a douche upon the back of the neck of alternating hot and cold water.

In subacute and chronic cases much aid can be obtained from inhaling compressed and exhaling into rarefied air, for then the respiratory movements are augmented and surprisingly large amounts of mucus emptied from the bronchi. Anodynes and anæsthetics are contraindicated except in the mildest and in chronic cases. In all severe acute cases they lessen cough and diminish the force of the respiratory acts. This increases the obstruction of the bronchioles, and cyanosis and pulmonary œdema are not unfrequently precipitated, when they should be avoided or guarded against.

Antipyretics are generally of no use. By lowering the temperature they do not remove the cause of its elevation. They contribute little to a feeling of well-being. They lessen the power of hæmoglobin to take up oxygen, and thus increase the desire already felt for it. Baths are useful, especially sponge-baths and douches, because of their stimulating effects upon the circulation and respiration, as well as for their antipyretic influence. They are not, however, needed unless the temperature is very high or the dyspnœa very great. They may then be resorted to frequently with advantage.

Some cases, mostly of the subacute type, are apparently the result of rheumatic conditions. The addition of salicylate of soda and of wine of colchicum to the usual treatment for bronchiolitis often effects convalescence with wonderful promptness.

In chronic cases the iodide of soda, continuously employed, proves useful in many instances, but generally does not effect a complete cure. In the acute cases nothing relieves the swelling of the mucous membrane so much as getting the bronchioles free from obstruction, so that the varying air-pressure which is caused by respiration may promote a more perfect lymphatic circulation, and thereby absorption of inflammatory exudates.

The frequent complications—emphysema and catarrhal pneumonia—will be treated of separately. (See pages 61 and 82.)

During convalescence the utmost care must be taken to avoid exposure to causes that might excite a new attack. Atmospheric changes must be guarded against by woolen clothing. A very careful and gradual exposure to air other than that of the sick-room must be contrived. Relapses can frequently be avoided, when recovery is fairly obtained, by a change of climate for a winter from the inclement and changeable air of northern temperate regions to localities warmer and more equable. During convalescence food can be varied and often can soon be made that of health, as the appetite returns and the digestive function is restored. When there is an atonic or depressed physical condition prior to the attack of bronchitis, and occasionally in other cases, the appetite does not return as the bronchitis lessens. Under these circumstances, bitter tonics, such as gentian or weak solutions of strychnia, especially when combined with active preparations of pepsin and with hydrochloric acid, are useful.

The general indications for the treatment of *chronic bronchitis* are the same as those for acute simple bronchitis; but each variety affords additional important indications. 1. For example, it is frequently necessary to forcibly maintain permeability of the air-passages, in order to overcome the dyspnœa which develops from the accumulation of secretions within the bronchi and from the thickening of their walls. 2. Less frequently, true asthma is present, and must be treated. 3. To modify purulent and fetid secretions is another frequent indication for treatment.

What I have termed chronic winter cough, usually the mildest and least harmful form of chronic bronchitis, must be treated in the main as is acute bronchitis. This form of the disease can often, in the first year or two of its course, be cured by prophylactic measures. A change of climate to a warm and equable region is usually necessary. The general health should be maintained at the highest degree of vigor. Exposing occupations and unwholesome places of residence should be avoided. Whenever acute exacerbations occur they should be promptly allayed by the usual medicinal treatment for acute bronchitis.

In some of the more chronic cases, especially when there is moderate but persistent dyspnœa on exertion, the iodides can be given with advantage. Their use should be continued for months; therefore, the iodide of soda is the preparation to be preferred. In children we obtain good results by the persistent use of cod-liver-oil. This is true especially of those who are slender and not well nourished. In adults the oil appears rarely to accomplish the same results, because it is taken in comparatively smaller quantities, and is more apt to cause disgust or digestive disturbances.

In dry bronchitis, with its usual paroxysmal cough, much relief is obtained from prolonged or permanent residence in a moist, warm climate, or by the frequent inhalation of steam vapor or atomized vapors. These lessen the frequency and severity of the cough, and, by adding moisture to the mucus within the bronchi, render it thin and more easily dislodged. Chloral is an efficient adjuvant to opiates to modify the spasmodic quality of the cough. It may be combined with ordinary anodyne and expectorant mixtures, and thus administered frequently in small doses (grm. 0.2 to 0.3); or, if the cough occur with much regularity it may best be administered in larger doses once or twice daily, a little before the paroxysm is expected to occur.

The persistent use of ammonium muriate or the iodide of ammonia or soda also increases the liquidity and usually the amount of secretions. I have often thought the most prompt effects were to be obtained from small doses often repeated, as, for instance, from 12 centigrammes (2 grains) of the muriate of ammonia every hour or two. After the secretions have thus been loosened, they can usually be maintained so by using larger doses two or three times daily. These remedies interfere somewhat with digestion and with the appetite, therefore they cannot always be efficiently employed in chronic cases. It is often more important to maintain good nutrition than to ease a cough.

Spasmodic dyspnœa, or true asthma, is to be relieved by the remedies whose use has been described (See page 12). Our efforts should be especially directed to the removal of the cause during the intervals of normal breathing.

Turpentine and similar drugs are especially adapted to those cases in which it is desirable to diminish the secretions of the bronchi or to render them less purulent. To this class of remedies belongs turpentine-oil, terebene, Venice turpentine, copaiba, eucalyptus, creasote (beech-wood), etc. It is often surprising how quickly a marked diminution in the amount of expectorate can be effected by these remedies. The secretions are lessened and made more tenacious. Therefore, when the expectorate is already tenacious and difficult to dislodge, the employment of these drugs makes expectoration more difficult and coughing harder and more prolonged. They should not be used under such conditions. They are especially indicated when the expectorate is thin and abundant.

The same drugs (especially turpentine and creasote) will convert, often with great rapidity, a thin, purulent secretion into one less abundant and muco-purulent. They certainly modify suppuration in the air-passages. Turpentine is best administered as an emulsion. Venice turpentine can conveniently be given in capsules, in doses of from 0.05 to 0.20 gramme (1 to 3 grains). Creasote is also conveniently administered in capsules, in doses gradually increased from 0.2 to 0.3 gramme (3 to 5 minims). Usually, these drugs must be combined with anodynes to allay cough.

The fermentation which characterizes putrid bronchitis is modified somewhat by the internal administration of remedies belonging to the group that has just been described, and most by creasote, eucalyptus, and terebene; but much more benefit is gotten from the employment of antiseptic inhalations. To cause antiseptics to penetrate to the deepest part of the air-passages they must be inhaled for hours at a time, and all the air that is breathed impregnated with them. This is best accomplished by using a respirator which covers the mouth and nostrils and can be worn for hours consecutively. The cotton or other absorbent which it holds and through which the inspired air is drawn must be saturated with carbolic acid, eucalyptus-oil, thymol, or similar volatile antiseptic. Steam inhalers and atomizers are sometimes employed, but are of comparatively little use. A Florence flask partly filled with hot water to which there has been added a volatile antiseptic, and so arranged that air can be drawn through the hot medicated water for inhalation, will prove moderately efficient; but as it cannot be used as continuously as the respirator, it is not to be preferred. By this treatment putrefactive changes can be greatly lessened and often entirely prevented. The best results are, of course, obtained in cases that come early under treatment.

Purulent and putrid bronchitis is usually accompanied by hectic fever and gradual but persistent loss of flesh and strength. Therefore we have, as prominent indications for treatment, the maintenance of strength. This is best accomplished by the systematic administration of the most nutritious food. Usually, the appetite is poor or capricious; therefore, food must often be varied. Exercise which is exhausting must be avoided. The fever, if it require any treatment, needs that which will be described (see page 159.) where tuberculous disease of the lungs is considered.

CHAPTER III.

BRONCHIECTASIS.

Anatomy.—Dilatation occurs most frequently in the medium-sized bronchi, rarely in the smaller ones, and still less frequently in the large ones. It occurs with equal frequency in both lungs, but oftener in the lower and middle lobes than in the upper. Usually a single bronchiectatic cavity is formed on one bronchus, but a number of them may be. They are then commonly separated from one another by unusually narrow tubes. The cavities vary much in shape. They may be fusiform or sacculated, round or angular. As a rule, they are not large; but they may occupy almost an entire lobe of the lung. They differ from other pulmonary cavities in that a single bronchus enters them, in that their walls show some of the anatomical elements of bronchial tubes, and in that usually they are not angular and are not intersected by bands of fibrous tissue and blood-vessels. The walls of the cavity are so modified by chronic inflammation that the anatomical elements characteristic of a bronchial wall are often obscured or, in places, obliterated. The mucous membrane varies in appearance, as it does in chronic bronchitis, when no dilatation exists. It may be unusually smooth and resemble a serous membrane, the cylindrical epithelium having been shed and replaced by flattened, pavement-like cells. Or it may be loose and thrown up into folds; or from it excrescences may protrude. Or the surface may be fenestrated by the atrophy of the muscular elements and persistence or hypertrophy of the elastic fibres. The bronchial tubes are often narrowed just beyond the cavities or obliterated. The latter change will cause collapse of the lung-tissue continuous with them.

Causes.—Bronchiectasis is always a secondary lesion. It usually results from chronic bronchitis, but may result from chronic inflammation of the lung or pleura. The bronchi are weakened by chronic inflammation, especially by ulceration, and therefore become dilatable. Increased air-pressure, which is caused by coughing, helps to produce their distension. Destruction of lung-tissue alongside of a bronchus also weakens its wall. Many sacculated cavities are due to ulceration and destruction of a part of a bronchial wall and the neighboring lung-tissue. The walls of such cavities are, for the most part, composed of granulation tissue. Tubercular ulceration of a bronchus is a common cause of cavities.

Interstitial pneumonia and extensive peribronchitis are lesions in the course of whose development new fibrous tissue is formed in extensive bands. Contraction of the newly-produced tissue always occurs in these bands. This will cause traction on the bronchus which is surrounded by the new growth, and will tend to dilate it. Large cavities are not made in this way unless, at the same time, adhesions have occurred between the costal and visceral pleura, which make the thoracic wall a fixed, unyielding point, from which the contracting tissue can pull. Cavities produced in this way are usually fusiform, but are sometimes angular. Chronic pleurisy, causing adhesion and the development of masses of connective tissue in the lung, beneath the pleura, sometimes produces bronchiectasis in a similar way.

Symptoms.—As bronchiectasis is always a secondary lesion, its symptoms must be expected superimposed upon those of the primary one. Often cavities of small size or deeply located cannot be discovered during life. The characteristic physical signs of bronchiectasis are those of a pulmonary cavity. They can be best described in connection with tubercular diseases of the lungs. (See page 133.) Bronchiectatic cavities occur both in non-tubercular and tubercular troubles. If the cavity is large, and especially if the secretions which it contains are thin, it is usually emptied periodically by coughing, and large amounts of expectorate are voided. A cavity can frequently be best emptied if a patient lies upon one side rather than the other, or assumes some position which enables the bronchus to drain it with thoroughness. To determine whether it is tubercular or not, one must ascertain the precise character of the primary affection.

Bronchiectatic cavities, if well drained by a bronchus, may remain open and undergo little change during many years; or they may gradually increase in size and destroy the lung extensively. A bronchus may become permanently obstructed. The cavity is thus converted into a cyst. Its liquid contents may then be absorbed and its more solid contents transformed into cheesy or, finally, into calcareous matter. A contraction and diminution in the size of a cavity frequently occurs, especially when it is converted into a cyst by obliteration of the bronchus; but it may also occur whenever unusual air-pressure is not produced within it or accumulating secretions cease to distend it. A perfect restoration of a dilated bronchus to its natural shape and size never takes place.

The chronic inflammation within a cavity can be treated by drugs, as chronic bronchitis always is. There is no specific medicinal treatment for it. The objects should be to prevent increased air-pressure within the lungs, the accumulation of secretions in the cavities, and the extension of ulceration. The first of these is best accomplished by lessening or preventing coughing, and the second and third by medication which will lessen the formation of secretions and of suppuration.

DISEASES OF THE LUNGS.

CHAPTER IV.

EMPHYSEMA.

Anatomy.—Emphysema begins with a dilatation of infundibular passages. As this increases the alveoli enlarge, their walls are stretched and finally torn. The infundibulum and alveoli are thus converted into a single small cavity, which by gradual inflation becomes spheroidal. Pin-head-sized sacks are thus formed. If the stretching of the lung-tissue persist or increase, the elastic fibres in the infundibular walls atrophy and the walls rupture, and neighboring sacks are thus made to communicate with one another and unitedly form larger cavities. It is rare that the cavities are larger than a pea or bean, but they may in exceptional cases be much larger. These anatomical changes are necessarily accompanied by destruction of capillaries. At first the capillaries covering the alveoli are stretched and narrowed. Later they are torn and destroyed. The vascularity of the emphysematous tissue is greatly lessened, so that it is pale or slightly rosy. The vascular changes lead to less and less perfect nutrition of the tissue, which in consequence becomes weaker and more easily stretches and tears. Prolonged stretching of the elastic fibres leads to a loss of elasticity in them. The dilated alveoli therefore have gradually less tendency to contract strongly, and to assume a natural form, and to fulfil their function of emptying the lungs of air that has been utilized in them.

The lesion of emphysema may be unilateral or bilateral in distribution, circumscribed or diffuse. The anterior, median, and lower borders of the lungs are oftenest affected, and the upper lobes oftener than the lower. The deep lung-tissue is rarely emphysematous. The lesion is almost confined to the superficial portion.

Emphysema, when it is extensive or generalized, makes the lungs unusually voluminous. Distension of the anterior and lower borders causes the lung to cover more or less completely the pericardium, to separate it from the chest-wall, and to depress the heart and liver. When the thoracic cavity is opened emphysematous lungs do not collapse. An incision in them will, however, permit the air to escape. The surface is unusually dry. The lungs when compressed creak little or none. From the cut ends of bronchial tubes muco-purulent matter can be expressed, for bronchitis more or less severe and extensive accompanies emphysema. Other lung-lesions are frequently present, as emphysema is a secondary one. When the enlarged lungs crowd the heart downward, the diaphragm may be simultaneously depressed; therefore, the liver and abdominal viscera may be pushed downward.

Causes.—It is probable that emphysema can be produced in several ways. If a lung is not properly nourished it becomes weak and emphysema is easily developed. This by many observers is claimed to be the most important factor in forming the lesion. When emphysema is generalized, as in old age, it undoubtedly is the most potent factor. When it is circumscribed it is chiefly the result of influences acting mechanically. For example, in capillary bronchitis the muco-purulent plugs which may obstruct the bronchioles permit air to enter the infundibula and alveoli, but as the expiratory act is less forceful than the inspiratory they do not permit them to be emptied. The alveoli thus gradually become distended. When emphysema is compensatory it must also be the result of mechanical influences only.

Emphysema is of very frequent occurrence in the aged. In them the lungs are not increased in volume, but the alveoli are distended. It occurs oftenest in men. It is observed occasionally in successive generations of a family, but this is probably a coincidence, and not evidence of its heredity. Severe coughing is a common cause of it. By coughing the air within the lungs is placed under unusual pressure, which of necessity stretches the lung-tissue. If coughing is frequent, chronic, and severe, it is especially apt to produce emphysema. The severe cough of pertussis or of chronic bronchitis may cause it. Small areas of emphysema are not uncommon in phthisis. Bronchiolitis, particularly if it is subacute or chronic, is especially liable to cause it. If one lung or one lobe is compressed or consolidated and rendered useless, the opposite lung or neighboring lung-tissue will become distended and emphysematous.

Symptoms.—If emphysema is circumscribed, often it cannot be detected by examination of the chest before death. If, as frequently happens, the anterior and lower borders only are distended, no subjective symptoms may be produced, but physical examination will reveal a diminished area of cardiac dullness, depression of the liver, absence of apex-beat and distant cardiac sounds.

If, however, the emphysema is generalized, the physical signs, as well as the subjective symptoms, are striking and distinctive. The thorax is abnormally enlarged. Its lateral diameter is greater than is natural.

The antero-posterior diameter is increased less. The spine is arched backward and the sternum forward. The centre of the thorax especially is distended. This gives to the entire chest a barrel-shaped appearance. The intercostal spaces are constantly stretched, and the whole thorax maintains permanently the position of deep inspiration. The acts of inspiration and expiration are performed by lifting and lowering the whole chest by the unusual muscles of respiration. The unusual work thus given to the sterno-cleido-mastoid, and other respiratory muscles which are rarely used, causes their hypertrophy.

Vocal fremitus is diminished, especially in advanced life, when the thorax becomes rigid and the costal cartilages ossified. On percussion, the area of pulmonary resonance is found to be permanently increased. The area of cardiac dullness is diminished or absent. The lower borders of the lungs remain distended, and the line of resonance does not move with the respiratory movements as it does in health. Vesicular murmurs are feeble or wanting. In many cases the crowing and piping sounds, which are caused by the coincident bronchial inflammation, obscure all others.

The apex-beat of the heart is invisible and usually cannot be felt. The cardiac sounds are not as loud as normal, especially at the apex. The second sound over the pulmonary orifice is accentuated in cases of chronic emphysema. This is due to obstruction to the pulmonary circulation which the destruction of capillaries causes. Cardiac murmurs are rarely developed, but if they exist they are due to dilatation of the heart.

The most prominent subjective symptom is dyspnœa. It is wanting in mild cases and when the lesion is circumscribed but is often very great. The vital capacity of the lungs is greatly lessened; therefore, less oxygen is furnished to the blood. The destruction of pulmonary capillaries also interferes with the blood's aëration. These are the important causes of dyspnœa. The expiratory power is greatly lessened, because of the immobility of the ribs and loss of lung elasticity.

Whenever dyspnœa is great cyanosis develops. It is rare that the causes of dyspnœa just mentioned are sufficient to produce much cyanosis; but, if bronchiolitis is extensive and the tubes much obstructed, it is common; or, if the heart-muscle become weak and passive engorgement develops, it is very evident. Death usually is caused by œdema of the lungs or heart-weakness.

The disease has an indefinite duration. If not severe it does not much interfere with life-work, but when severe causes chronic invalidism. In itself it rarely leads to fatal results, though it increases very greatly the danger to life when other diseases attack its victims. The prognosis, therefore, depends upon the nature and severity of accompanying diseases.

Treatment.—The causes of emphysema should be removed whenever possible, and if it has not been of too long standing recovery then becomes probable. Many cases of bronchitis which cause it are curable. Compression of the lung from pleural effusions, which provoke emphysema of the opposite lung, is often capable of relief. Many other causes of it are removable.

The treatment of chronic cases must embrace a careful regulation of habits, so that as good general health can be maintained as possible. Tonics, such as strychnia and quinine and iron, are invaluable whenever tissue enfeeblement or degenerative changes are prominent factors in the causation or maintenance of the lesion.

They must be employed for long periods of time. The best results are usually obtained by occasional changes in the preparations which are used. Iodides do much good when chronic bronchiolitis is the cause of the emphysema.

Chronic emphysema of long standing results in an absolute loss of elasticity in the affected portions of the lungs, and a degree of alveolar and capillary destruction which is irreparable. In cases less in degree and duration the elasticity can largely be restored by training the lungs to contract and expand. This can be accomplished by pneumatic apparatuses of various kinds. Exhalation into rarefied air will cause partial collapse of the alveoli by withdrawing from the lungs an unusually large amount of their contents. By repeated exhalations of this kind the lungs are made forcibly to contract and expand to a normal extent, and gradually a habit of approximately normal respiration can be acquired. Very great temporary relief to dyspnœa can always be obtained in this way. As the loss of lung elasticity is largely the cause of the prolonged distension, considerable relief is often obtained by wearing continuously a broad elastic band about the thorax. It gradually increases the force and deepens expiration, and thus enlarges the vital capacity of the chest.

CHAPTER V.

ATELECTASIS.

Anatomy.—The part of the lung which is collapsed is usually depressed below the surface of the rest, is angular, uneven, and brownish or bluish red. If the atelectasis is of long duration, it may be grayish in color. An entire lung may be collapsed, but in most cases a part only is affected. The atelectatic portion is hard, and feels fibrous when the lesion is chronic. It is leathery or brittle. It does not crepitate. If compressed, no air can be squeezed from the cut surface, although mucous generally can be from the bronchi. The affected tissue sinks in water. At first, the alveoli appear smaller than normal, more angular, and sometimes quite flattened. The capillaries are usually visible as swollen and tortuous vessels. Later on, the epithelial cells are loosened from the alveolar walls, and degenerate. The connective tissue is thickened by hyperplasia of its cells. The capillaries become less swollen, and the outlines of the alveoli less evident, because more and more collapsed and contracted. On account of these cirrhotic changes the capillaries are less permeable, and the increased blood-tension thereby produced in the pulmonary artery leads, in extensive and chronic cases, to hypertrophy of the right ventricle.

Cause.—Atelectasis is commonly caused in four ways: 1. It is congenital. The affected lung or portion is then uninflated. This condition may gradually be corrected, or remain a permanent one. It occurs oftenest in premature, prolonged, or difficult births.

2. It is due to absorption of the air from the alveoli. This occurs oftenest when the small bronchi are obstructed by mucous plugs, the result of bronchiolitis. Fresh air cannot enter the obstructed tube, and, therefore, the air that is held in the unventilated alveoli is slowly absorbed by the blood. The oxygen is first taken up, and then the carbonic-acid gas and, later, the nitrogen. Obstruction of large bronchi by swollen lymph-glands, tumors, etc., which rarely occurs, is also followed by absorption atelectasis. 3. Compression atelectasis is usually due to pleuritic effusion, pneumothorax, tumor of the pleura and lung, rarely to pericardial effusion, dilated heart, or aneurisms. Other causes are: mediastinal growths, deformity of the thorax, abdominal tumors, and ascites. 4. The last form is known as marasmatic atelectasis. It is the result of the great debility which is produced by wasting diseases, such as tuberculosis and typhoid fever.

Congenital atelectasis usually occurs in the base of the lungs; not very unfrequently in the anterior lower borders, and rarely at the apices. Absorption and marasmatic atelectasis also occurs oftenest in the lower part of the lungs. The part affected in compression atelectasis depends entirely upon the location and character of the cause.

Symptoms.—If congenital atelectasis is extensive, the child will breathe superficially, and with unusual rapidity. Often a soft, murmuring noise is produced by respiration. The child will refuse the breast. Its face will be gray or livid, the pulse quick and weak. Death may result from suffocation. Twitchings and even convulsions often precede the termination of life. If the collapsed areas are extensive, the lung and even the thorax may be retracted, but they are rarely of sufficient size to cause dullness on percussion, bronchial breathing, or other signs of consolidation.

In acquired atelectasis the symptoms are the same in kind, but they are frequently transitory, as compensatory emphysema usually accompanies them. When collapse first occurs, fine crepitant râles can be heard. If the air has only been partly removed from the affected parts, tympanitic resonance may be audible. If the air is altogether absorbed, and the area is at least one and one-half inches in surface-area and two-thirds of an inch in thickness, dullness can be detected, and broncho-vesicular or in larger areas bronchial breathing and increased vocal fremitus can be heard. If the conditions are present which lead to hypertrophy of the right ventricle, the second sound over the pulmonary artery will be accentuated. Percussion may demonstrate enlargement of the heart to the right, and substernal pulsations will be evident.

Treatment.—If the cause of atelectasis can be removed, deep breathing, pulmonary gymnastics or inhalations of compressed air may, singly or combined, re-expand the collapsed tissues, but if the collapse has lasted long enough to have caused cirrhotic changes and obliteration of the alveoli, recovery is impossible. If the atelectatic areas are not too large, emphysema may fully compensate for them.

CHAPTER VI.

Hæmorrhagic Infarction.

Causes and Anatomy.—Hæmorrhagic infarcts in the lungs are possible, because the pulmonary arterioles are terminal. An embolus, which will produce them, may be derived either from the veins of the body or from the right side of the heart. Fibrinous clots are not uncommonly formed in the right ventricle when it is dilated, and in the veins when they are inflamed. The infarcts may form in any part of the lung, but they are oftenest observed near the surface and in the lower part.

If an embolus obstruct an arteriole, the circulation will cease beyond it. The pressure is, therefore, nothing in the artery beyond the obstruction. The blood now flows back into the arterioles from the capillaries, and even from the veins, and engorges the area supplied by the obstructed artery. The blood extravasating into the pulmonary tissue and alveoli consolidates the lung at this point. The solid mass is conoidal, with an apex at the point of embolism and base usually at the pleura. Infarcts vary in size from a cherry-stone to a hen's egg, and rarely are larger. They can usually be seen through the pleura as dark, purplish, slightly raised masses, which feel firm to the touch. The pleural surface is usually congested and covered with more or less fibrin. When cut through, the conical shape of an infarct becomes evident. At first its surface is purplish red. If resolution take place it becomes reddish brown, a rusty color, or even grayish, with an excess of brownish-black pigment. Under the microscope there is seen at first only a mass of blood in the alveoli and lung-tissue. If resolution occur, white cells become more numerous and the red ones disintegrate, and thereby form pigment granules, which are deposited in the interstitial tissues or absorbed into it. The interstitial tissue is thickened and a permanent toughening and pigmentation of the lung results. The air once more enters the air-cells, but they expand and contract imperfectly, because of their thick walls. The arterial embolus may also disappear, but usually it leaves a thickening of the artery's wall at the point of embolism. The permanent pigmentation gives the cirrhosed tissue a brown or slate color. If resolution is delayed, and especially if the infarct is large, a part of the lung-tissue may be destroyed, being liquefied and converted into an odorless, brown pulp. This either finds its way into a bronchus and is expectorated, or it is absorbed and the cavity obliterated by cicatrization. In rare cases the cells and the tissue involved in the infarct disintegrate slowly, dry, and are transformed into a caseous and cretaceous mass, which is surrounded by a fibrous capsule. If the embolus contain pyogenic matter an abscess is the result. Pleuritic adhesions may form over infarcts.

An infarct is not caused whenever there is an embolism of a pulmonary artery. Sometimes death occurs before it can be produced. Sometimes collateral circulation by the capillaries or communicating arteries may prevent it.

Symptoms.—Infarcts may exist and produce no symptoms. Embolism of the large arteries may cause sudden death, or when this does not happen sudden and great dyspnœa and thoracic oppression may be felt. The most characteristic symptom is hæmoptysis. The expectorate may be mixed blood and mucus, or consist almost entirely of dark blood. The hæmorrhage may last a few hours only or several days. Pleuritic pains may also be felt. If the infarct is small or deeply seated, physical examination will afford no positive information. Pleuritic friction-sounds are sometimes heard. If the infarct is larger and superficial it may produce an area of relative dullness on percussion, and crepitant râles or bronchial respiratory sounds may be audible over it. Fever may be absent or present. A diagnosis will depend upon the existence of a cause for embolism of the lungs and upon the occurrence of hæmoptysis. The prognosis depends upon the primary disease, the strength of the patient, and character of the embolus. If the malady complicates heart disease it is rather unfavorable, for it signifies weakness of the right ventricle.

Treatment.—When the causes of embolism of the lung exist, bodily rest is essential for its prevention. Treatment must be symptomatic. Pleuritic pains may necessitate the use of anodynes. The primary disease requires special treatment. Resolution is best assured by removing the primary disease and maintaining a good circulation and good general nutrition.

CHAPTER VII.

HYPOSTATIC AND PASSIVE CONGESTION.

Causes of Hypostatic Congestion.—Hypostatic congestion is a secondary lesion of frequent occurrence. It develops when the venous circulation through the lungs is impeded by an enfeebled heart's action, and when, through the prolonged retention of one position, the blood stagnates in the veins. It is the prolonged retention of the recumbent posture that usually causes it to develop in the posterior and lower part of the lungs. The enfeebled heart is oftenest the result of wasting illness, such as typhoid fever and suppuration. Fractures and paralysis may also cause the essential weakness and dorsal decubitus. Impediments to respiration, such as pleuritic adhesions and thoracic deformity, or compression of the lungs by distension of the abdomen, increase the tendency to hypostatic congestion.

The capillaries and veins are distended, and impart to the affected tissue a purplish and often almost a black hue. The alveoli are filled with serum. A few blood-corpuscles find their way into them. The epithelial cells are cast off and become granular. If the lesion is quite persistent the alveoli contain large numbers of these cells, and closely resemble those consolidated by catarrhal pneumonia. Under such conditions the lungs become heavy and firm. They do not crepitate. Atelectasis and pulmonary œdema are often associated with hypostatic congestion.

Symptoms.—This lesion may persist for days or weeks, or disappear in a few hours. It is readily overlooked if attention is concentrated too closely on the primary disease. In diseases in which it often occurs (especially typhoid fever) it should be guarded against and watched for. Frequently rapid respiration is the only symptom which suggests its existence. If the lesion is extensive it may cause cyanosis. Cough is not often present; when it is it may be accompanied by a mucous, muco-purulent, or even purulent expectorate. Fever is not caused by hypostatic congestion unless inflammation supervenes. Percussion over the posterior thoracic surface will usually at first give a tympanitic resonance, because of the relaxed condition of the lung. In more chronic cases various degrees of dullness exist. At first fine, moist râles are heard, and the respiratory sounds are often feeble. Later the sounds become bronchial and consonant. Increased vocal fremitus is then demonstrable. These physical signs are those of consolidation of the lung, and, therefore, the same as those of pneumonia. It is to be distinguished from the latter disease chiefly by the history of its development, its secondary character, and by its usual bilateral distribution. Œdema rarely produces consolidation, and usually causes the râles to be more widely diffused. A positive diagnosis is at times impossible. The prognosis is grave, though by no means hopeless.

Treatment.—The most successful treatment is prophylactic. It consists in frequently shifting the patient from side to side, and thus preventing the gravitation of blood to any one part of the lungs; and in administering a cardiac tonic, such as digitalis. In febrile cases spongings and baths maintain a better peripheral circulation, by creating a greater degree of arterial tone, in consequence of which a better balance is maintained between the arterial and the venous blood. When the lesion is once established the same care must be maintained to prevent its increase. But the carbonate and chloride of ammonium are now useful, both as cardiac stimulants and as expectorants, if there is an accompanying bronchitis. Counter-irritants will temporarily relieve the congestion when it first occurs.

Causes and Anatomy of Passive Congestion.—Brown induration, or chronic venous hyperæmia, is difficult to diagnose with certainty. It may be suspected whenever there is much dyspnœa accompanying heart diseases. It is, however, only one of several factors causative of dyspnœa. When the lungs are examined, the pleural surface is usually, at least in places, reddish purple, and the interlobular septa are evident, because they are pigmented by a dark-brown coloring matter. Generally slight emphysema exists here and there. The lungs feel hard and dense, especially in places, and mostly about their bases. The cut surface permits a reddish-brown fluid, mixed with air, to exude. It is redder than normal, though not bright, but a brick-red. The pleura and interlobular septa are unusually thick and contain much of the red pigment. Under the microscope the alveolar walls are seen to be unusually thick. The capillaries are enormously distended and very tortuous. Their walls are thick. The blood-corpuscles completely fill them. In the alveoli there are many red corpuscles and large cells, which contain large granules of golden-brown pigment. In the alveolar walls the same pigment can be seen, but usually it is a little darker in color. Where the lungs are most dense the alveoli are most filled with such cellular contents.

This lesion is developed by a venous stasis, which, slowly dilating the veins and then the capillaries, causes finally a moderately copious serous exudation in the alveoli, and some hæmorrhage. It is thus that the red corpuscles and serum are expressed into the alveoli. The epithelium is soon detached and almost wholly lost. The white corpuscles take up the pigment-granules which are formed by the disintegration of the red cells. They increase in size, and thus form the large pigmented cells that have been described as a part of the contents of the alveoli. Some of the pigmented cells find their way into the lymph-channels and deposit in them their coloring matter Minute hæmorrhages from which pigment is formed occur in the pleura and interstitial tissues. The small bronchi are greatly congested. The walls of the distended vessels are thickened. The mucous membrane is somewhat swollen, and many epithelial cells are loosened.

Symptoms.—The changes in the bronchi and congestion of the alveolar vessels, which diminishes the air-spaces in the lungs, are important factors in producing dyspnœa. The partial filling of the alveoli with serum and cells diminishes the lung-capacity. The slow and imperfect pulmonary circulation causes a lessened oxidation of the blood, and also contributes to produce dyspnœa.

Anything that obstructs the pulmonary vein may cause this lesion. All forms of valvular disease of the heart may do it, but oftenest it is a mitral stenosis that does. It is a peculiar and not well-explained fact, that in one case brown induration will be developed to the fullest extent, and in another, with apparently the same causes and conditions present, it will not exist. Often, in these cases, exaggerated or puerile respiratory sounds are heard during life. If the large pigmented cells can be found in the sputa, a positive diagnosis may be made. Red blood-cells are also sometimes seen in the sputa. The pigmented and blood cells rarely color the expectorate.

The prognosis depends upon the primary heart-lesion. Treatment must be addressed entirely to the latter.

CHAPTER VIII.

PULMONARY ŒDEMA.

Anatomy.—The anatomical changes which are characteristic of œdema of the lungs are enlargement of them and increase in weight. They are often very pale in color, but they may be congested. They do not collapse when the thoracic cavity is opened. When pressed between the fingers pits remain, as they do in other œdematous tissue. When a lung is incised an abundance of serous fluid flows from it, which is usually colorless, but may be either pink or red, according to the degree of congestion that exists. The fluid may or may not be frothy. When only a part of a lung is involved in œdema, it is the most dependent part. If circumscribed inflammation of the lung—as croupous pneumonia—exist, it is often found to be bordered by a zone of œdematous tissue.

Symptoms.—The symptoms of œdema of the lungs which are of diagnostic value are not developed unless the œdema is considerable in degree. Œdema is often the immediate cause of death. It may develop with great rapidity and produce fatal results in a few hours, or may develop more gradually. When œdema is the outgrowth of other lesions of the lungs, pathognomonic symptoms are often wanting. Œdema of the lungs does not cause fever.

Persistent dyspnœa is one of the most striking symptoms which it produces. As it becomes greater, cyanosis develops. The lips and finger-nails become purplish, and the skin ashen, usually cool, and bathed with perspiration. Coughing occurs with more or less frequency, and considerable or, sometimes, very large quantities of serous fluid are expectorated, which may or may not be frothy, pink or colorless. The abundant expectoration usually does not continue long, for, as the lungs become more and more completely filled and the blood cyanotic, mental dullness develops, which gradually passes into somnolence. This hebetude causes a cessation or diminution in the frequency and strength of the cough. Under such circumstances the fluid gathers in the throat and causes a coarse rattle there. Respiratory movements become more and more shallow, then a little irregular, and finally they cease.

If a physical examination of the chest is made, the respiratory movements will be observed to be rapid and shallow. Unless the œdema is due to a localized inflammation which is in one lung only, the respiratory change will be found alike on both sides of the chest. This symmetrical distribution of physical signs is especially characteristic of the œdema which accompanies heart, renal, and general diseases. Palpation reveals no abnormality. Percussion reveals a normal resonance, or, frequently, a semi-tympanitic resonance. Moist râles are abundant. At first, they are fine; later, coarse. A diagnosis is possible only when we find dyspnœa, cyanosis, an abundant serous expectorate, and a cause for œdema.

Causes.—Rarely, cases of œdema of the lungs are observed which develop rapidly, and for which no adequate explanation can be given. They are sometimes termed idiopathic œdema of the lungs, serous apoplexy, or serous pneumonia. Exposure to cold is an alleged cause, but we have not positive proof that it is one.

Localized inflammations are a cause of circumscribed œdema, which occasionally spreads and involves an entire lung, or both of them. This happens not infrequently in cases of crupous pneumonia, and œdema then becomes the immediate cause of death. Often pulmonary œdema is a part of general œdema, such as complicates heart and renal diseases. Mitral-valve disease is especially apt to lead to pulmonary œdema, but all cardiac diseases that are accompanied by dilatation of the heart and weakness of it are liable to precipitate œdema of the lungs. The cardiac weakness which results from prolonged fever or other wasting disease is frequently the cause of pulmonary œdema. The lesion is also caused by the paralysis of the left side of the heart, which occasionally occurs just prior to death. We find, then, upon post-mortem examination, œdema of the lungs, although no symptoms of it existed prior to death. When it accompanies renal affections, it is due in part to an enfeebled circulation and in part to an impoverishment of the blood.

Treatment.—The most successful treatment is prophylactic, and should be used when causes of œdema exist. When the lesion is established, especially if it involve the lungs extensively, it is rarely amenable to treatment. A prognosis in such cases must be guarded, and the immediate danger to life recognized.

Prophylactic treatment must vary with the cause which exists. In fevers œdema can be avoided by frequent changes of position which will prevent hypostatic engorgement. Cardiac tonics, such as digitalis and strychnia, should be administered when the heart is feeble and the blood-vessels relaxed, to counteract these conditions. Frequent spongings of the surface of the body stimulate the circulation, and maintain a better tone in the peripheral vessels. It is also an important aid in maintaining a good general circulation.

In cardiac and renal diseases, when general œdema is extensive and pulmonary œdema must be anticipated, diaphoresis, diuresis, and catharsis are useful modes of treatment, as they lessen the general œdema. Heart tonics are usually necessary.

In the so-called idiopathic cases venesection has been found to do temporary good. It should be followed by the administration of digitalis, strychnia, and ergot,—agents which will maintain a vigorous action of the heart and a good degree of vascular tone.

If œdema of the lungs must be treated after it has developed, reliance must be placed upon digitalis, strychnia, and ergot. I have seen such good results obtained from the inhalation of oxygen in these cases that I believe it should always be tried if the gas can be obtained. During the last winter I saw, in consultation, a woman just delivered of a child and simultaneously attacked by pneumonia. In a few hours extensive œdema of the lungs developed and threatened immediate destruction of life. She labored severely for breath, was cyanotic, covered with cold perspiration, and almost pulseless. Oxygen was administered by inhalation for several minutes every half-hour. The cyanosis disappeared, the labored breathing lessened, the skin became warm, and the pulse full, firm, and steady. So prompt and decided was the improvement that the treatment was persevered in and life undoubtedly prolonged for several days. Oxygen inhalations will undoubtedly enable us to save some cases that would otherwise prove fatal, by maintaining life for a few hours or days, until a turning-point in the primary disease can be reached.

CHAPTER IX.

CATARRHAL PNEUMONIA.

Causes.—This disease may be acute, subacute, or chronic in its course. It is secondary to others; most commonly to bronchiolitis. It is a frequent complication of infectious fevers. Measles, diphtheria, influenza, and whooping-cough are complicated by it with especial frequency. It is rare except in childhood and old age. Debility predisposes to it. It is one of the usual lesions in chronic tubercular diseases of the lung. Pneumonic nodules are often built up around miliary tubercles.

Anatomy.—Catarrhal pneumonia is characterized by the development of solid nodules which may be scattered through the lung. They vary in size from a pinhead to a walnut. Large nodules are produced by the coalescence of the smaller. In number they may be few or almost countless. They are most frequently formed along the posterior part of a lung, and are more numerous at the base and gradually less toward the apex. When they exist the surface of the lung is not quite even. The superficial nodules cause depressions in it. Over the nodules there is often pleuritic inflammation. They can be readily felt as hard, compact bodies. If a section is made through one it appears purplish in the early stages and grayish yellow later on. The surface is dry and granular. They contain no air, but a yellowish or brownish fluid can be scraped from them. Around the nodules the lung is often slightly emphysematous. A bronchiole forms the centre and focus of each nodule.

From it a muco-purulent plug can be squeezed. Many suppose that the inflammation which causes the nodules originates from drawing into the lung detritus and pus from inflamed bronchi; others believe that the inflammation extends by continuity from the bronchioles to the lung. The latter seems the most probable explanation, although the lesion may at times be produced in both ways.

The central bronchiole in each nodule will be found to be filled with desquamated epithelium, granular matter, pus-cells, and mucus. Its wall will be found thickened and infiltrated with round cells. The adjoining alveolar walls are similarly affected. Toward the margin of the nodule the alveolar walls are less and less thickened. At first the alveoli are filled with serum and the capillaries engorged, but soon the alveolar epithelium becomes granular, is cast off, and helps to fill the air-cell. Leucocytes are also abundant, especially in the alveoli nearest to the central bronchus. Occasionally red corpuscles may be seen forming part of the contents of the air-spaces. They are usually observed in the earlier stages. In the air-spaces nearest to the central bronchus, fibrin is also observable when consolidation first occurs. In the alveoli at the periphery of the nodule the consolidation is due almost entirely to the abundant and closely-packed, large, oval, epithelial cells which fill them. In these alveoli the process is purely a catarrhal one, while nearer the central bronchus the inflammation is accompanied by round-cell infiltration.

As catarrhal pneumonia is almost uniformly a complication of some form of bronchitis, the anatomical changes within the bronchi, characteristic of their inflammation, are to be expected coincidently. The pneumonic nodules may undergo resolution. The contents of the alveoli will be absorbed, and complete restoration may take place. Instead of resolution occasionally the contents of the alveoli become dry, the cells degenerate; finally, the alveolar walls degenerate, and the whole is transformed into a fine, hard, dry, grayish, cheesy mass, which may remain unchanged indefinitely.

Symptoms.—A positive diagnosis is often impossible. Catarrhal pneumonia is so uniformly a complication that it is frequently obscured by the primary disease. It is rare that it is ushered in by a chill or any other noticeable phenomenon. When it develops, the symptoms of the primary bronchitis are usually intensified. Cough is generally drier, often more painful, and occasionally accompanied by slight pleuritic stitches. Respiration is more rapid. In severe cases dyspnœa is marked, and cyanosis may develop. General prostration is great. The temperature is higher than in simple bronchitis. In the latter it rarely rises to 103° F., while in catarrhal pneumonia it often exceeds this. It follows no definite type, but is usually remittent, the morning temperature being nearly or quite normal. Deffervescence takes place slowly. In children, breathing is often painful, and accompanied by moaning. The thorax is tender. No characteristic physical signs are developed unless the nodules coalesce into patches of at least two inches superficial area and of two-thirds of an inch depth. Then the usual evidences of consolidated tissue can be found; increased vocal fremitus, bronchial respiration, and dullness being the most important ones. The lesions are usually discoverable in both lungs at the same time.

A probable diagnosis can be made in the course of bronchitis if the temperature is abnormally high, the breathing unusually quick, and the prostration much greater than is to be expected from acute bronchitis.

If the chest is tender, and pleuritic stitches are felt during breathing or coughing, the probability is still greater. A certainty in the diagnosis can be felt if, in addition to these symptoms, patches of consolidation can be demonstrated.

The disease often runs a very acute course, lasting only a few hours, or two or three days. Frequently it pursues a subacute course, and may last for two or three weeks. When it accompanies measles, it is inclined to be acute; when whooping-cough, subacute.

Treatment.—The treatment is the same as would be employed in capillary bronchitis. More attention must be paid to the maintenance of strength. Food in its simplest forms must be administered regularly. The heart's strength must be preserved by the administration of digitalis, strophanthus, and similar tonics. The carbonate of ammonia, camphor, and other diffusible stimulants are required when the heart flags. The rules for their administration are practically the same as in treatment of croupous pneumonia. (See page 101.) To stimulate deeper breathing when it is shallow and cyanosis is developing, aid can be derived from douches and sponging of the skin of the body, especially with alternating hot and cold water. Fomentations so prepared as to envelop the whole chest often relieve dyspnœa and cough, and contribute to the well being of the sufferer.

The prognosis must always be guarded, and the disease looked upon as dangerous. It is very fatal; from one to two-thirds of all cases die.

CHAPTER X.

CROUPOUS PNEUMONIA.

Nature and Causes.—Croupous pneumonia may be defined as a fibrinous inflammation of the lungs, affecting simultaneously large areas and accompanied by fever. The exact nature and mode of causation of this disease form unsettled questions. It resembles closely an infectious disease, and without much doubt is one. As in other infectious diseases, its general symptoms are not correlated with the local inflammation. For instance, the fever, often the delirium, and the rapidity of respiration cease suddenly, and before consolidation has disappeared from the lungs. It also resembles them in that it occurs epidemically and endemically. It has rarely been found to be the cause of death in infants who are born while their mothers are suffering from it. There is also much, though not conclusive, evidence that micro-organisms are its exciting causes. Several micro-organisms are capable of producing fibrinous inflammation of the lungs. No one form is found to uniformly accompany pneumonia. The pneumococcus of Friedländer was the first form carefully studied, but it is not observed as often as Fraenkel's coccus. Seibert believes that the latter is the usual cause of sthenic pneumonia, or of those cases that run the classical course, and that the former is the common cause of those which run a prolonged and more typhoid course. Eichhorst thinks that secondary pneumonias are not the result of superimposed infection, but of the infectious agent of the primary disease. For example, that in typhoid fever and measles, the cause of these diseases is the cause of the pneumonia which may complicate them. It must be admitted, however, that as yet our knowledge of the relationship of micro-organisms to croupous pneumonia is not definite. It is probable that the cases now known as croupous pneumonia constitute what from an etiological stand-point might be regarded as several distinct diseases. The micro-organisms supposed to cause croupous pneumonia are frequently found in the pleural, pericardial, and meningeal exudates that result from complicating inflammations.

Pneumonia resembles a general disease, in that its course is cyclical, that its general symptoms are not correlated to the local ones, and that it is endemic and epidemic. The general symptoms are undoubtedly due to a poisoning of the system by some chemical substance or substances produced by the local inflammation of the lungs or the micro-organism that causes it. In this respect there is a strong analogy between the disease and typhoid fever. (See Physiolog. Act. Typhoid Poison, etc.) What may be the nature of the poison or poisons is not known. The recent researches of Roger and Gaume upon the toxicity of the urine open the way to a better knowledge. They found that during the period of pneumonic fever the urine contained only a third or fourth as much of toxic matter as is normal, and that at the time of crisis it suddenly augmented to more than normal, or at least to the normal amount. This would suggest that the poison which produces the general symptoms is not eliminated for a time, and that, when it is, they cease. The toxic ingredients of the urine are certainly not any of its well-known constituents.

Anything that lessens vitality predisposes to the disease. Bad hygiene and debility from other kinds of illness are predisposing causes. Excessive use of alcohol not only increases the gravity of the disease, but greatly augments an individual's susceptibility to it.

Pneumonia occurs in all parts of the world. The statement has been generally repeated since Drake first made it, that it is most prevalent in the southern third of this country, and least in the northern third. By a recent review of statistics, N. S. Davis, Sr., has found that it is most prevalent in the middle third and least in the southern.

It varies much in prevalence at different seasons. In large cities it is endemic, although, at least in the northern cities, primary croupous pneumonia is rare in summer. In Chicago, and other cities with a similar climate, it is most prevalent from December to March, and much the most prevalent in December and January. Some years it is epidemic. Its severity also varies from year to year. It is most prevalent during or immediately following intense and penetrating cold weather. A moist, cold air will provoke an increase of all kinds of respiratory diseases.

Individuals in every period of life are liable to attacks of croupous pneumonia. It is, however, most prevalent during youth and the first half of manhood, and is rare in infancy. It is commoner among men than women. It has been claimed to be contagious, but observations are not numerous enough as yet to prove that it is so. Although often exposure to cold is apparently a cause, in very many cases such exposure cannot be traced. It is a disease, like bronchitis, erysipelas, and rheumatism, that strongly predisposes to renewed attacks. Wounds that can be regarded as causative occur in a very small proportion of cases. I have a few times known an injury to cause pleurisy and a slight pulmonary hæmorrhage, which was followed, in two or three days, by pneumonia. The injured lung-tissue was undoubtedly the focus of the pneumonic inflammation.

Anatomy.—Either of the lungs and any part of them may be affected. Usually an entire lobe or lung is involved. The lower lobe upon the right side is most frequently the seat of pneumonic inflammation. Not unfrequently one lobe or one lung after the other is attacked, and, therefore, upon the post-mortem table we find different parts of the lungs exhibiting simultaneously the characteristic appearance of two or more stages of pneumonic inflammation. Anatomically, four stages of the disease are recognizable: a stage of (1) congestion, (2) red hepatization, (3) gray hepatization, and (4) resolution.

In the stage of *congestion* the portion of the lungs affected is enlarged, heavier than natural, but light enough to float on water. The pleural surface is reddened. Upon it the congested vessels are often visible as red lines. The lung crepitates and pits somewhat, when compressed. The cut surface is red, and a pinkish and often more bloody, frothy fluid flows from it. The alveoli are partly filled with fluid and air. Under the microscope the epithelium is seen in places to be loosened, and some of the cells lie in the alveoli, and undoubtedly float in their fluid contents. Red and white corpuscles are also more or less numerous. They are least numerous at the beginning. The capillaries are distended, tortuous, and crowded with corpuscles.

In the stage of *red hepatization* the affected parts are firm and hard, still more enlarged, and two or three times heavier than normal. The solidified lung sinks when placed in water. The pleura is usually red, though it may be uniformly pale after death. It is covered with serum, which makes it feel to the touch as if covered with soapy water, and usually with flakes or patches of fibrin. The lung is so much enlarged that it completely fills the thorax, and its surface is furrowed by the ribs. When removed, it looks like a cast of the cavity. If compressed, the lung does not crepitate. It is brittle. The cut surface is dry, granular, and dark red. The portion of the lung immediately adjoining the solidified part is, to a greater or less extent, œdematous. The opposite lung or other lobe of the affected lung may appear perfectly normal, or present evidences of bronchitis. From the bronchi in the consolidated part fibrinous casts can be drawn, which, when floated upon water, exhibit the outline of the bronchioles. The larger bronchi are always more or less inflamed.

Under the microscope the alveolar walls of the solidified lung are seen to be thickened and the capillaries on them congested. The air-spaces are compactly filled with small round-cells. About the margins of the alveoli desquamated, large, oval, epithelial cells can be seen, and here and there a few red corpuscles. All these cells are held together by a mesh-work of fibrin-fibres. The pleura also seems thickened. On its surface there are white cells, occasionally red ones, and some fibrin.

The stage of *gray hepatization* is looked upon by a few pathologists as occurring only in fatal cases. Often a part of the solidified lung will be in this stage and a part in the former, which causes the whole to appear mottled-gray and red. In size, weight, and solidity it resembles the lesion of the preceding stage. It is, however, more brittle and easily torn. The pleural surface is covered more or less abundantly with a fibrinous exudate, and is still furrowed by the ribs. The cut surface is reddish yellow, or, later and more characteristically, yellowish gray, dry, and even more granular than in the preceding stage. Under the microscope the alveolar walls still appear thickened from cellular and serous infiltration. The lymph-channels are distended with granular material and cells, which have been absorbed from the air-spaces. The capillaries are no longer distended. The cells which fill the alveoli are more granular, fattily degenerated, and some of them are disintegrated. The fibrin is in shorter threads, and rarely attached to the alveolar wall. The contents of the alveolus appear more concentrated in the centre and detached from the walls.

In the stage of *resolution* the lung is soft and flabby. The pleura may still show evidences of inflammation, or present a normal appearance. A pus-like fluid exudes from the alveoli exposed upon a cut surface. Under the microscope the air-spaces are seen to be filled with numerous cells, which resemble pus-cells, and an abundance of granular matter, which is cellular *débris*. The alveolar walls are thickened chiefly by distension of the lymph-channels, which are crowded with absorbed cells and granular material. The cells which in the earlier stages of the disease fill the alveoli and solidify them during resolution disintegrate or migrate into the lymph-channels and are absorbed. A part of the mass may be expectorated when it loosens.

The kidneys, liver, and intestinal tract are usually congested,—at least, so long as the solidified lung impedes the circulation. The kidneys and liver often undergo albuminoid infiltration. After death the right cardiac cavities are usually full of blood and postmortem clots, and the left are empty. The muscles of the body are, as a rule, flabby and soft, and some of the fibres may undergo waxy degeneration. Venous congestion of the brain is usual.

Rarely a case of croupous pneumonia is met with in which the lesion becomes *chronic*. Instead of resolution taking place and the fibrinous clot originally filling the alveoli undergoing solution and absorption, it becomes organized. The round-cells which fill it are gradually transformed into connective-tissue cells. The air-spaces are thus permanently obliterated. As the connective tissue is developed contraction occurs, and the solidified lung grows smaller and hard. Often bronchiectatic cavities are formed in these cases. Tuberculosis, abscess, and gangrene are other lesions that develop occasionally in pneumonically-inflamed tissues.

Symptoms.—Different cases of croupous pneumonia vary in the development and duration of their symptoms. A majority, however, follow a typical course. In the typical cases there are rarely prodromal symptoms. Malaise may be felt for one or two days preceding the attack. It is usually ushered in by a severe chill of considerable duration. Almost at once the sufferer feels that he is very ill. During the day following the chill, and the two or three next, the patient develops characteristic symptoms. The face looks full and flushed. The skin is dry and hot. The pulse is quick, full, and firm. The temperature commonly is from 103° to 104° F. Soon after the initial chill pain is felt, in the region of the nipple, upon the affected side. It is aggravated greatly by coughing or deep breathing. Although coughing occurs, it is suppressed as much as possible, because of the pain. The respirations are from 30 to 40 per minute. They are short or superficial. The patient lies upon his back or the affected side. His tongue is covered, at first, by a white coat, which soon becomes brownish and dry. Appetite is wanting. Thirst is considerable. The bowels are constipated. Delirium often exists. He is usually extremely ill. In favorable cases, at the end of the fifth or seventh day, or, exceptionally, by the ninth or thirteenth day, the fever suddenly disappears. Simultaneously the other distressing symptoms lessen or disappear. The breathing becomes much less rapid. The pulse becomes almost or quite normal. The mind is clear. The skin is cool. A copious sweat often accompanies the subsidence of the fever. Coughing persists. Sometimes the side is still painful, but usually it is not. The appetite improves. Digestion seems more perfect, and thirst is no longer felt.

In unfavorable cases, on the fifth or sixth day the delirium becomes duller. A semi-comatose or somnolent condition develops. The pulse grows soft, smaller, and quicker. By degrees the skin grows cool and ashen in color, and is covered with a clammy sweat. These changes are first noticed in the extremities. Involuntary discharges from the bowels and bladder are usual. The respiratory movements become more and more shallow and labored. The nares dilate, and the larynx is lifted with each inspiration. Death oftenest occurs on the sixth, eighth, or ninth day. Its immediate cause is usually either heart-failure or suffocation from œdema of the lungs.

In the course of the disease the physical signs—first of congestion, and later of consolidation, of the lungs—develop. Usually the earliest signs of a lung-lesion are detected on the second day of the illness, but their appearance may be delayed to the third or fourth day.

In a considerable number of cases the fever does not leave suddenly, but gradually subsides by lysis. In secondary pneumonias there is usually no chill at the inception of the attack, and all the symptoms are obscured by those of the primary disease. The existence of pneumonia is suspected only from the unusual rapidity of respiration, and proved by physical examination.

The sideache, which is an early and in many cases a bothersome symptom, is not always present. In cases of secondary pneumonia it is generally wanting. The pain varies much in degree. It is usually stitch-like and dull simultaneously. It is aggravated by deep breathing and coughing. Often there is some tenderness to the touch. The pain is usually felt about the nipple, or a little outside of it, on the affected side. The pain is the result, chiefly, if not wholly, of pleurisy. That a neuralgia may accompany the pleuritic pain is possible, for in rare cases pain is not felt upon the side on which the lung is inflamed, but upon the opposite side.

Coughing is an early and almost constant symptom. In primary pneumonia it is usually severe. It is suppressed, as far as it can be, because of the pain which it excites. In secondary pneumonia, and especially when the primary disease causes mental stupor,—such as exists in typhoid fever, for instance,—the cough may be absent. An expectorate may be wanting in children and aged persons: in the one because they do not know how to expectorate, and in the other because they are too feeble. In others weakness or the failure to cough occasionally prevents expectoration; as a rule, it is present. At first the sputa is scant and adhesive. It is often expelled from the mouth with difficulty. As soon as the blood and fibrinous exudate has formed in the lung the sputa becomes red; it is a dull, brick red. The color is produced by red blood-corpuscles in it, but also, and chiefly, by blood-coloring matter in solution. In a small proportion of cases the sputa is purplish red and likened to "prune-juice." In a considerable number of cases the sputa is not raised from the lungs, but from the larger bronchi, and is not red. In most instances in which the sputa is abundant there can be found in it small gray or yellowish-gray balls, which, if dropped upon water and shaken, will unfold and reveal themselves as branching fibrinous casts of the smaller bronchi. Rarely, these are composed of the spiral threads which so uniformly form bronchial casts in asthma. The sputa contains mucus-corpuscles, some cells that resemble those of pus, red blood-cells, and a few epithelial cells. Micro-organisms of various kinds can be found, especially the cocci supposed to be peculiar to pneumonia. Gradually the sputa loses its red color, becomes purulent and loose, then frothy, and finally it ceases.

Respiration is always abnormally quick. It is shallow, especially upon the affected side. The rate of respiration is abnormal, in that it does not correspond to the pulse-rate or temperature. In health and in other fevers the pulse bears about the ratio to the respiration of 4½ to 1, but in pneumonia it is nearly 2 to 1. The number of respirations is commonly 45 per minute, and may be 60 or more. This peculiarity of the respiration-pulse ratio is almost pathognomonic. The rapidity of breathing is, in part, due to the pleurisy, which prevents deep respiration because of the pain. It is also quick, because of the increased temperature of the blood. Something else must also excite the respiration, for it is quick, out of proportion to the temperature, even when pleuritic pains are not felt. The diminution of lung-space does not account for the quickened breathing; for, after crisis, when the temperature has fallen and the pain has ceased, the respiratory movements, though quicker than normal, are one-half or two-thirds less than earlier in the illness. It is probable that respiration is quickened, at least partly, by a poison which acts upon the nervous system, and is produced in the course of pneumonic inflammation.

The pulse bears about the usual ratio to the increase of temperature. It is quick, usually from 100 to 120, and at first full and strong. In favorable cases it remains full and strong; in unfavorable cases it grows soft and small and quicker. An intermittent pulse is an unfavorable symptom, and is due to a complication or feebleness of the heart. An abnormally slow pulse usually indicates disease of the brain.

The temperature varies in its range and course. It is rarely more than 105 degrees or less than 102 degrees. In typical cases it pursues a continuous course until crisis occurs, on the fifth to the ninth day. At the onset of the disease the temperature rises rapidly, and in twelve hours is usually 103 degrees. Its highest point is generally reached on the third day; on the fifth, seventh, or ninth the temperature falls to normal. Before the crisis the daily range is about 1 degree. In most instances the temperature falls during the night of the fifth day, and thereafter remains normal. Crisis is sometimes less abrupt: for instance, during the fifth night the temperature will fall from 104.5° to 102° F.; during the sixth day it will rise to 102.5° F., and during that night fall to normal and remain there. False crises may also occur; most frequently they happen upon the third day. The temperature may fall to normal, but usually only to 100° F. or thereabouts. If such a crisis occur earlier than the fifth day it is rarely permanent. The typical or classical cases of pneumonia convalesce with a crisis, but a considerable proportion of cases convalesce, after a slow subsidence of temperature, by lysis. The whole temperature-curve then closely resembles that of typhoid fever, but its course is usually shorter.

During the height of the disease the urine is scant in quantity, red, and often cloudy. Its specific gravity is increased. The relative proportion of urea that it contains is greater than natural. Sodium chloride is almost or quite wanting. Occasionally, small amounts of albumen can be found in it. If nephritis does not follow or complicate the pneumonia the albuminuria ceases when the fever subsides.

The signs elicited by a physical examination are all important for diagnostic purposes. In the stage of *congestion* the respiratory movements appear to be deficient upon the affected side. This is partly due to the congestion and obstruction of air-cells, and partly to the pain which full expansion would cause. Vocal fremitus over the affected parts is normal or increased. Resonance is normal, or sometimes, just before consolidation occurs, semi-tympanitic. Auscultation reveals the most characteristic sign,—fine crepitant râles.

In the stages of *consolidation* the chest over the affected area usually seems slightly distended, and is almost motionless. Vocal fremitus is always increased. There is dullness over the consolidated parts; it is extensive, usually covering an entire lobe, or a large part of one. The area of dullness is frequently bordered by a narrow area that is semi-tympanitic; over the remainder of the lung the resonance may be normal. The respiratory sounds are bronchial whenever there is consolidation. An abnormal degree of resistance is often felt by the hand, when pressed upon the side, or when percussion is being practiced.

As *resolution* progresses dullness gradually lessens, or is replaced temporarily by semi-tympanitic percussion-sounds, which are due to the relaxed condition of the lung-tissue. Fremitus gradually becomes normal. Coarse, moist râles are usually present. If there is much bronchitis similar râles may be heard in the stage of consolidation. The distension of the side ceases and the respiratory movements become more normal.

As different portions of the lungs may be successively involved, we may be able to demonstrate resolution in one part and consolidation in another. Pleurisy, with effusion, complicates a few cases. Pericarditis and endocarditis are rarer complications. Meningitis is an occasional, and usually a fatal, complication.

Often dual names, such as typho-pneumonia, are applied to individual cases descriptive of complications. The following can be recognized as more distinct varieties: *Protracted pneumonia* is the form in which the fever disappears by lysis. *Spreading pneumonia* is the form in which the lesion gradually increases by involving neighboring tissue in successive invasions. *Wandering pneumonia* is the form in which different, but not contiguous, parts of the lungs are involved successively. *Relapsing pneumonia* is the form in which, after crisis has occurred and convalescence has apparently been established, the lung again becomes consolidated, either where it first was or at some other point. *Intermittent pneumonia* is a name applied to rare cases which are probably complicated by malaria; all the symptoms of the stage of congestion will suddenly develop, persist for three or four hours, and then disappear, leaving the patient apparently well. The next day, or the second day, a similar attack will occur. Several of these threatenings of pneumonia may occur, each a little more intense than the preceding one, and at last a full development of the disease will take place. When established, such a case may follow the classical type of the disease.

So-called *chronic pneumonia* follows the acute attacks. Sometimes fever persists for weeks, and even months, but it is hectic in type. Often, in such cases, an abscess forms, or tubercular complications develop. In other cases the fever ceases, and a part of the patient's strength slowly returns; but he remains short-winded, often coughs slightly, and fills the affected side less than the opposite. The affected side is a little dull, and increased fremitus persists there. The respiratory sounds may be consonant, but more frequently they are low and broncho-vesicular. The thorax on that side slowly contracts. The opposite lung becomes emphysematous, is hyperresonant, and the respiratory sounds in it are exaggerated. Resolution may finally take place, but often the lung is permanently crippled.

Diagnosis.—In most cases a direct diagnosis can be made from the sudden onset of the characteristic symptoms; from the course of the fever; from the occurrence of rusty sputa; from the characteristic pulse-respiration ratio; and from physical signs.

It can be distinguished from dry pleurisy by the greater severity of the disease, by the pulse-respiration ratio, by the rusty sputa, and the physical signs of lung congestion or consolidation. It can be distinguished from typhoid fever by the absence of abdominal symptoms and of the typhoid rash; by the presence of the usual pulse-respiration ratio; often of a bloody sputa, and the physical signs of lung consolidation. It is more difficult to distinguish between hypostatic congestion and the catarrhal consolidation which frequently accompanies it, and a complicating croupous pneumonia. If fibrinous casts are found in the sputa they are quite characteristic of croupous pneumonia. The congestion is usually bilateral, and involves the dependent parts of both lobes of the lungs, while croupous pneumonia is usually unilateral, and involves only one lobe,—generally the lower. Hypostatic congestion does not cause as great physical prostration as does a complicating pneumonia.

Treatment.—There is no specific treatment for croupous pneumonia. It must be treated symptomatically. The mildest cases require good nursing, and almost no medication. The severe cases require a supporting and stimulating treatment. The disease is especially characterized by great prostration, which, in some cases, might almost be called shock. For its relief, as well as to allay the pleuritic pains which are usually at first felt, opiates must be employed. When pneumonia is of moderate severity, and occurs in one who is robust, Dover's powder is a useful preparation. If, however, the patient is debilitated at the start, the opiate should be combined with quinine. A pill of morphia and sulphate of quinine is then the most conveniently administered. The opiates should be used in such doses and repeated with such frequency as to insure at least moderate relief from pain, but, if possible, constant drowsiness should not be caused. When quinine is used, from 10 to 12 grains daily is sufficient.

In the stage of congestion depleting agents rarely will shorten the course of the disease, or even prevent consolidation. Venesection has most frequently accomplished this. It can be safely practiced upon sthenic cases, but is almost invariably useless for those who are not vigorous and robust when attacked by the disease. Instead of venesection, especially in asthenic cases, blisters, dry cups, and fomentations on the affected side are indicated. Aconite and veratrum have been commended in this stage, because, by relaxing the peripheral vessels, a depletion is produced. They must be used with caution, however, and only in the stage of congestion, for they tend to weaken the heart's action. There is so much danger to life in this disease, because of cardiac weakness, that its strength must always be preserved. After consolidation has taken place, the ammonium carbonate (grm. 0.18 to 0.3—gr. iij to v) or mild chloride of mercury (grm. 0.015 to 0.06—gr. $\frac{1}{4}$ to j) may be given to hasten the solution of the exudate.

The severest cases must be treated like the milder ones, but there are two great dangers to life which must be guarded against. The first is heart-failure, and the second is suffocation. In order to maintain the heart's vigor, resort must be had to cardiac tonics and diffusible stimulants. The best of the latter group of remedies is ammonium carbonate. In order to get its full effects as a cardiac stimulant, it must be taken in solution, for its irritation of the gustatory nerves reflexly excites the heart to more vehement action. After absorption it also stimulates it. The effect of ammonia is very transitory. It must, therefore, be often repeated. The doses may be from grm. 0.18 to 0.3 (gr. iij to v) every hour. A solution 1 part of camphor in 10 of olive-oil has been used, with excellent effect, by hypodermatic administration. Diffusible stimulants are chiefly indicated when failure is imminent. When the impulse of the heart first loses its forcefulness and the pulse begins to soften or to grow small, cardiac tonics, such as digitalis and strophanthus, are indicated. The latter is to be preferred, for it provokes quite as forceful contraction of the heart as digitalis, and does not contract the peripheral vessels to the same extent. The latter effect should be avoided, because it increases the work of the heart. The tinctures of strophanthus and digitalis are the most certain preparations for administration. I have found 10 minims of each, repeated every four or three hours, the best dose for steady use, although, at times, a little larger or a little smaller dose may be preferable. In many severe cases it is necessary to give those remedies almost from the beginning of the disease.

Spongings of the body, showers, and douches, especially when applied about the chest and the back of the neck, are strong cardiac and respiratory stimulants. They also are chiefly indicated when heart-failure is imminent.

While these especial cardiac stimulants and tonics are useful as prods or goads to the flagging organ, it must not be forgotten that the strength which they excite is a temporary one. They call forth, as it were, the organ's reserve strength. To give true strength to the heart reliance must be placed upon food. Strychnia and quinine are often serviceable, because they prompt a more rapid and better degree of general nutrition. They can be advantageously administered in combination with the cardiac tonics.

Suffocation generally results from pulmonary œdema. I need not repeat the treatment which is indicated when it occurs. (See page 81.) If pulmonary œdema exist digitalis is to be preferred to strophanthus, because it does contract the vessels, and may possibly hinder exudation through their walls. Atropia, ergot, and strychnia are the other drugs upon which reliance must be placed. The inhalation of oxygen will save a number of otherwise fatal cases.

When bronchitis is severe or precedes pneumonia it greatly increases the danger to life, as it increases the tendency to pulmonary œdema. Opiates must be used with caution in these cases, for, by suppressing cough, they prevent the clearing of the air-tubes, and they also tend to dilate the peripheral vessels, which may hasten œdema.

Antipyretics, especially antipyrin and its congeners, have been extensively used in pneumonia, but with doubtful efficacy. The temperature is rarely so high that, in itself, it is a source of danger; therefore, it is not necessary to lower it in order to save life. Antipyrin, acetanilid, and similar drugs depress the nervous system, frequently increase the depression or condition of shock which the disease produces, and may even produce a state of collapse. They lessen the oxygen-carrying power of the blood. In many cases it is very important to preserve this power. To depress the temperature does not shorten the course of the fever or modify the important local lesions. The statistics of cases treated with antipyretics show often an increased mortality, and certainly not a greater ratio of lives saved. I have discontinued their use in these cases.

Quinine is especially indicated in atonic cases, and then is best given in daily doses of 10 or 12 grains. When malarial troubles complicate the pneumonia larger doses may be required.

Alcoholics have been used in pneumonia with great freedom. They have been prescribed for two purposes, neither of which, I believe, is attained by them. They have been used as foods and as diffusible stimulants.

Whether they are foods or not is a question still debated. It is proven, however, that those who take them cannot accomplish as much work or endure as much hardship as those who do not. They are not, therefore, strength-giving. As diffusible stimulants, therapeutists state that they provoke stronger pulsations of the heart when given in small amounts. If their anæsthetic effects are produced they weaken the heart's action. Even in small doses no stimulation is produced in persons who are accustomed to them. Such effects are very transitory; and, if the drug is frequently repeated in order to maintain them, its anæsthetic properties are manifest. In large amounts,—several ounces daily,—as it is often prescribed, it lessens the oxygen-carrying powers of the blood, and thus delays tissue change. The drug is not necessary for the treatment of this disease, as abundant clinical evidence has demonstrated to me. In Mercy Hospital, during the last ten years the death-rate from pneumonia has been a little less than 12 per cent., though no alcohol was employed in the treatment of the cases. During the same time the death-rate in other Chicago hospitals, where alcohol was used with greater or less freedom, was 28 to 38 per cent. I have no hesitation in discarding alcohol in the treatment of pneumonia. I believe that often it has done harm.

Pneumonia commonly runs a short but severe course. Very great prostration may be felt when convalescence is first established, but usually strength is rapidly recovered. However, those who were feeble when attacked by the malady may recover slowly. It is important in all cases, but especially in the latter group, to maintain strength during the illness. As an inclination for food is usually wanting, it must be taken as a necessity. It should be given in a form as concentrated and as easily digested as possible. Milk is most uniformly the best diet. A little should be administered every hour or two during the severest portion of the illness. It will be least liable to disturb the stomach or cause repugnance if it is thus administered. If the heart is weak, beef-tea, or chicken-broth, or strong coffee should also be given. They are stimulants rather than foods. During convalescence food should be simple in character, but should be gradually varied as the appetite returns.

In chronic pneumonia we must combat a tendency to abscess formation, or treat abscesses, if they exist. To check the inflammation, blisters or sometimes milder counter-irritants, occasionally applied, are useful. Anodynes are needed to allay cough, when it persists. Strength must be conserved and, if possible, improved by careful feeding. If the fever disappear, the tendency of the affected side to contract and to prevent the full inflation of the lung must be counteracted by respiratory gymnastics. At first, gentle, enforced, deep breathing, and, later, the pneumatic cabinet, or, better still, a residence for a few months in high altitudes, should be tried. The respiratory gymnastics expand the lungs, help to lessen their congestion, and prevent permanent contraction of the affected side. The dry air of high altitudes also promotes a more rapid absorption of inflammatory exudates. If an irregular remitting fever persist, which, although not enough to keep the patient bedridden, is enough to make high altitudes dangerous, a residence at low altitudes, in dry climates, —such as can be found in Western Texas, Arizona, and Southern California,—is exceedingly beneficial. Usually such a change improves the appetite, tempts to more active exercise, to the rapid absorption of the exudate, and, with these changes, to the disappearance of temperature. If abscesses or tubercular disease supervene, they must be treated as they would be under other circumstances. The medical treatment of the chronic cases must be symptomatic and tonic. Iron and bitter tonics must usually be given.

Pneumonia is very frequently a fatal disease. The proportion of fatal cases has been variously estimated in different hospitals and by different observers. It varies also from year to year. The mortality is estimated as high as 25 per cent., in some hospitals. In my own hospital and private practice during the last five years, it has varied from 4 to 12 per cent. It is especially fatal in persons more than 40 years of age. In children and in the earlier years of manhood recovery occurs in a very large proportion of cases. In those who are addicted to the free use of alcoholics the mortality is much greater than in total abstainers. Croupous pneumonia of the lung's apex is especially dangerous, for it is usually accompanied and followed by tuberculosis, if it does not at once cause death. Complications always greatly increase the danger to life. A temperature of more than 105 degrees must usually be regarded as significant of danger. A temperature of 104 degrees accompanies the disease in its moderately severe form. It is found, also, that an increase of temperature after the fourth day is usually significant of an unfavorable result.

CHAPTER XI.

CIRRHOSIS OF THE LUNG.

Anatomy.—Cirrhosis of the lung is often called interstitial pneumonia. It results from the formation of large amounts of new connective tissue, which begins with infiltration of the interstitial tissue of the lungs by embryonic connective-tissue cells. The change may involve the whole of the lungs, and is then very moderate in degree. It may be very circumscribed, or limited to a single lobe, or more frequently to a part of a lobe. When the lesion is extensive it causes contraction of the lung, so that, for instance, an upper lobe may be transformed into a small mass of firm, tough, unaërated tissue not one-quarter its normal bulk. More frequently smaller areas are involved, which produce deformities of the lungs and render portions of them useless for respiratory purposes. If the cirrhotic tissue is cut into, it often creaks like cartilage. Usually the pleural cavity over cirrhotic masses is obliterated by adhesions. If a section is made into a lung containing a cirrhotic area, the diseased portion appears as a band, or more or less extensive area of gray, compact, hard, and fibrous tissue. About it the air-spaces are irregularly shaped and have thick walls, which deform them. As the cirrhotic mass enlarges the interstitial tissue becomes thicker and thicker, and the air-space is diminished proportionately and finally is obliterated. Pigment is often deposited, in considerable amounts, in the cirrhotic tissue, and produces a mottling of the cut surface. Bronchial tubes in this tissue are often dilated (see page 57). Less frequently they are obstructed or obliterated by the contracting scar-tissue. Adhesive pleuritis and contracting cirrhotic lung-tissue often produce a deformity of the thorax, and impede breathing by drawing together the ribs; narrowing and making immobile the intercostal spaces. Adhesions, the results of cirrhotic changes in the lungs, often occur between the lungs and pericardium, and result in displacement of the heart. Whenever the cirrhotic change is extensive, hypertrophy of the right ventricle occurs in order to compensate for the obstruction to the pulmonary circulation offered by the obliteration of many capillaries and small arteries.

Symptoms.—The lesion is always secondary, and the only appreciable signs of diagnostic value that it produces are physical ones. Small or deeply-seated areas of cirrhosis may exist and may not be demonstrable before death. Larger areas, and especially superficial cirrhosis, cause retraction of a portion of the thoracic wall, which is visible to even a carelessly-observing eye. This retraction occurs most frequently about the apices, as it causes the flattening of the chest in consumption. It less frequently produces deformities about the lower part of the thorax. The chest is always diminished in size by these changes, as well as rendered irregular in shape. The ribs are drawn together and comparatively immovable, so that respiration is not equal upon both sides of the thorax, and is especially limited at the points of retraction. The other physical signs are those of consolidation of lung-tissue. Vocal fremitus over the area of consolidation is increased, and often bronchial fremitus can be detected. Dullness is noticeable at the same point. It is usually relative. Its completeness depends upon the size and superficial position of the solid, cirrhotic tissue.

Broncho-vesicular or bronchial respiratory sounds are usually heard over the same area. Vesicular sounds are always obscure, or wanting. If dilatation of a bronchus has occurred, tubular or cavernous sounds may be audible.

The demonstration of displacements of the heart, liver, or viscera adjoining the lungs or thorax is excellent corroborative evidence of the existence of cirrhosis. If the vascular obstruction is considerable in the lungs, the second cardiac sound at the pulmonary orifice will be accentuated. When the right ventricle is enlarged and hypertrophied, its increased size can be demonstrated by percussion, and infra-sternal pulsations can be felt.

Causes.—Cirrhosis often occurs primarily in old age. It is then moderate in degree and generalized. Few of the air-cells are completely obliterated, but most are misshapen, contracted, and surrounded by thick, unyielding walls.

In all other cases it is secondary and usually to chronic inflammations of the pleura, lung, or bronchi. It is present, to a greater or less extent, in all cases of chronic phthisis.

Treatment.—The treatment consists, first, in obtaining a cure of the primary affection, or removal of the cause; second, in correcting the deformity that the lesion produces, or in compensating for it. Good hygiene, and especially the breathing of pure air, is essential to prevent fresh irritation of the lungs and exacerbations of the trouble. Pleurisies, bronchitis, and similar affections, which may be the cause of cirrhosis, are to be treated by customary methods. To prevent the formation of cirrhotic tissue, and even to cause its disappearance, the double chloride of gold and sodium, chloride of potassium, and iodide of potassium and sodium are commonly recommended. I have not been able to convince myself of their utility, except in one case, where syphilitic inflammation was the cause of pulmonary cirrhosis. In this case the iodides were beneficial.

To correct the deformities and displacements which cirrhotic tissue in the lungs causes, pulmonary gymnastics are of great utility. Deep breathing, frequently resorted to, will often suffice to cause again an expansion of a retracted area of the chest. It accomplishes this not by removing the cirrhosis, but by bringing into full use many neighboring air-cells that were before imperfectly inflated, because of the weakness of the respiratory act, and, to some extent, by producing a compensating emphysema adjoining the cirrhotic area. Often good results are quickest obtained by directing the deep inhalation to be taken while other parts of the lungs than the cirrhotic ones are compressed or kept unexpanded, so that as much air as possible can be forced into the affected areas. This mode of expanding the lungs is especially applicable when the lower part of one lung is affected. By bending the body to the unaffected side, and retaining the position while as deep an inhalation as possible is taken, the retracted part of the chest often can be more rapidly and perfectly re-expanded. When the upper part of the lungs is contracted and the thorax flattened, exercise of the pectoral muscles and others attached to the chest, shoulders, and arms aid deep breathing in expanding the crippled parts. Pneumatic differentiation and residence at high altitudes are especially useful to distend a contracted thorax.

Prognosis.—The prognosis of cirrhosis of the lung depends entirely upon its cause. If resulting from tubercular trouble, the prognosis is necessarily governed by the nature and progress of the disease. If the cause can be removed, the lesion ceases to extend, and mechanical treatment may prevent permanent deformity. It is impossible to remove the cirrhosis; but it may be compensated for by hypertrophy or dilatation of the lungs.

CHAPTER XII.

PULMONARY ABSCESS AND GANGRENE.

Causes of Abscess.—Abscess of the lung is not common. Pyogenic matter deposited in the lung is essential to its formation. Such matter may gain access to the pulmonary tissue with foreign bodies which lodge in the bronchi. It frequently produces an abscess when croupous or catarrhal pneumonia consolidates the parenchyma. Oftener it gains access to the lungs in septic emboli. They are produced in pyæmic conditions. Multiple pulmonary abscesses may then be formed. Penetrating wounds of the thorax also rarely admit it, and are causes of suppuration.

Anatomy of Abscesses.—Abscesses may be formed in either lung, and in any part of them, but they occur oftenest in the upper lobes. They vary greatly in size, —from almost microscopic dimensions to cavities which occupy an entire lobe. The cavity is usually globular, but may be angular. It contains pus. Its walls vary in thickness. Within they are composed of numerous layers of greenish-yellow or brownish pus, and outside of this of granulation and fibrous tissue. By suppuration the lung-tissue is destroyed and dissolved. Suppuration may occur in a lung consolidated by pneumonic inflammation or by hæmorrhage. Recovery may be produced by a spontaneous drainage and contraction of the cavity, and ultimate obliteration by granulation upon its inner surface. Very rarely, small abscesses may desiccate, contract, and be transformed into cheesy or calcareous nodules. More or less complete spontaneous drainage is the rule. It generally occurs into a bronchus, but may occur into a pleural or into the peritoneal cavity, where it excites purulent inflammation. Very rarely, an abscess ruptures through the thoracic wall.

Symptoms of Abscess.—The clinical history of pulmonary abscess varies with its origin. If it complicate pneumonia crisis is usually delayed, or is imperfect, and the fever soon rises again. Pains are felt in the chest, and often local bulging is recognizable. Finally, the lesion manifests itself by a copious expectoration of pus. Abscess is to be suspected if the patient has been addicted to alcoholics, or if the pneumonia has been a hæmorrhagic one, and a very purulent sputa is expelled.

The sputa constitutes the most characteristic symptom. It is usually abundant, and resembles laudable pus. It appears suddenly. If it is not expectorated freely it may become offensive in odor, but loses this characteristic when it is freely evacuated. In chronic cases it is rarely lumpy, like the expectorate of phthisis. If allowed to stand it separates into two layers,—a lower granular and an upper serous one. In the pus particles of lung-tissue, large enough to be recognized by the eye, are almost invariably present. Under the microscope the bits of lung are seen to consist of elastic fibres, which often display the outline of the alveoli. Besides the pus-cells, fat-crystals and reddish pigment-granules are frequently seen. Hæmatoidin-crystals are unusually abundant. Micro-organisms are numerous; they are chiefly round, and occur in groups.

Often, at first, the physical signs of pulmonary consolidation can be discovered, especially when the abscess is superficially located. Later, when it is drained, the signs of a cavity supersede these.

Fever, of a hectic type, is almost invariably present.

If perfect drainage is not established spontaneously or artificially a progressive loss of flesh and strength occurs, which finally ends in death.

Diagnosis of Abscess.—A diagnosis is based upon the history and the presence of the characteristic sputa. It must be differentiated from (1) abscess of neighboring organs,—as of the liver or spine,—which may penetrate the lungs and be drained by the bronchi. Bits of lung-tissue cannot be found in the sputa in these cases. From (2) phthisis, which is accomplished by the history, and by the presence of tubercle bacilli in the sputa of the latter; from (3) pulmonary gangrene by a purulent, and at least not constantly fetid, sputa, by the absence of mycotic plugs, and a larger number of hæmatoidin-crystals.

Causes of Gangrene.—As the occurrence of pulmonary abscess requires the presence in the lungs of pyogenic matter, so, for the production of gangrene, the bacteria of putrefaction must be present. It is probable that they frequently enter the lungs in small numbers by the bronchi, but they are unable to harm healthy tissue. Conditions of disease in the lungs, especially inflammations, make it possible for them to develop gangrene. Rarely, a healthy person may, by an accident, permit putrefiable matter to enter a lung,—as, for instance, particles of food; and with these as a nidus gangrene may certainly develop. It will oftenest develop thus in persons who are insane, delirious, comatose, or whose muscles of deglutition are paralyzed. At times bits of decomposing tissue, from cancers or ulcers about the mouth or throat, may be drawn into the bronchi and cause gangrene. Very rarely, ulcers accompanied by decomposition may penetrate the thoracic wall and attack the lung, causing putrefaction in it. Fetid bronchitis may also be the cause of the lesion. Pneumonia and tuberculosis are occasionally complicated by gangrene. In these cases the putrefaction occurs in the inflamed tissue, but is not the cause of it. Gangrene may be caused by emboli that have been carried from other parts of the body where decomposition is going on. For instance, they may be derived from extensive and foul bed-sores or abscesses.

Anatomy of Gangrene.—Either or both lungs may be attacked. The right one is oftenest. The lower lobes are more liable to invasion than the upper. The lungs may be affected diffusely and very extensively, or only small parts of them may be involved. Gangrene oftenest spreads from a single focus, but may originate from several. The affected tissue first becomes soft, and brownish or greenish black. It exhales an offensive, fetid odor. The tissue soon liquefies in part, and a ragged, irregular cavity is formed, which is filled with a greenish-black fluid and bits of decomposing lung. An area of catarrhal or croupous inflammation usually exists about this cavity. The cavities commonly rupture into the bronchi, but many open into the pleura, pericardium, peritoneum, or, very rarely, externally through the thoracic wall. The gangrenous process gradually involves more and more of the contiguous tissue. In favorable cases a circumscribing inflammation separates the necrosed tissue from the rest as a sequestrum, and forms a limiting wall around it. The latter may be thin or thick, and in time may become fibrous. Extensive hæmorrhage is rare, as it is prevented by coagulation within the pulmonary vessels before they are destroyed. If the cavity is thoroughly emptied of its putrefying contents, the fibrous capsule usually contracts it. The granulation tissue by which it is lined may cause its final obliteration. Pleurisy is a common complication of gangrene, and is often purulent.

Symptoms of Gangrene.—The symptoms which accompany gangrene vary much. As it is often secondary, the symptoms of the primary affection may obscure those that are due wholly to the gangrene. In such cases the sputa affords the most characteristic signs of the disease. It is often large in amount. It may be as much as fifteen to twenty ounces per diem. It resembles in physical attributes the sputa of fetid bronchitis. Its odor is extremely offensive. It taints the breath of the patient, and even the air about him for many feet. If allowed to stand it stratifies, as does the sputa of fetid bronchitis. In the lowermost layer pus-cells and granular matter predominate, but plugs and shreds of tissue can also be found in considerable amounts. The plugs contain numerous crystals of fatty acid imbedded in countless bacteria. But what distinguishes this from the sputa of fetid bronchitis is the particles of lung-tissue which can be found in it. Various chemical substances are produced by the putrefaction which are characteristic of it. In very rare cases upon the post-mortem table gangrene of the lungs is demonstrated, though never suspected before death, for the characteristic sputa and fœtor oris were wanting.

The other symptoms are not peculiar to it. Cough and pain in the side are usual. Dyspnœa is sometimes marked. Centrally-located gangrene does not modify the physical signs, but when it is superficial and extensive, at first, the signs of pulmonary consolidation, and, later, of pulmonary excavation are demonstrable. The signs of pleurisy are frequently present, and may bscure the others.

Fever usually exists, but, as a rule, it is quite irregular. If the gangrenous slough is freely eliminated from an encapsuled cavity and absorption is thus prevented, fever may be absent.

The fœtor of the breath often destroys the patient's appetite and may even cause vomiting. The absorption of putrefying matter rarely causes rheumatoid pains. Metastatic abscesses may result from gangrene. Death has often been immediately caused by a secondary abscess of the brain.

A diagnosis can only be made when the characteristic sputa is present.

Prognosis of Pulmonary Abscess and Gangrene.—The prognosis of pulmonary abscess must be a guarded one. It is always a grave disease. But a large proportion of the cases that occur are curable. A small proportion recover spontaneously. They are those in which the abscess is thoroughly drained by the bronchi or by rupture through the thoracic walls.

Diffuse gangrene of the lungs is rarely recovered from. Circumscribed gangrene may be. It must always be remembered, however, that infection of other parts of the lungs may occur so long as any gangrene remains. Death is the result either of general loss of strength or of complications, such as pulmonary hæmorrhage, pleurisy, and brain-abscess.

Both in abscess and gangrene of the lungs the prognosis will depend, in part, also, upon the vigor of the patient or the existence of other underlying disease.

Treatment of Abscess and Gangrene.—Gangrene may be prevented by removing food from the air-passages if it accidentally fall into them when the muscles of deglutition are partly paralyzed, or a patient is mentally dull.

Persons who are thus liable to errors of swallowing should be closely watched while eating.

In abscess, if the sputa is offensive, and always in gangrene, much benefit can be derived from the inhalation of antiseptics. The best effects are obtained by the prolonged inhalation of them. Therefore, antiseptics are best administered by a respirator, which should be worn for hours. Turpentine, oil of pine, eucalyptus, beech-wood creasote, and carbolic acid are among the most useful antiseptics. Inhalation through the respirator may at first cause a feeling of oppression, but this will pass off as the user becomes accustomed to it. Simpler means of inhalation, though less certainly useful, may be employed. For instance, the vapors of turpentine or of creasote, or of eucalyptus, may be inhaled from a pitcher in which they have been mixed with steaming water, by fitting a paper cover over the pitcher for a mouth-piece. Or a Florence flask with an air-inlet and inhaling-tube may be employed. When such antiseptics are inhaled they not only check the putrefaction, but also check suppuration. The prolonged use of the mask or respirator insures the impregnation of all the air in the lungs by them.

Symptomatic treatment may be needed to relieve pleuritic pains, or to allay nausea and vomiting. The radical treatment for abscess and gangrene of the lungs is surgical. Success can usually be expected from it. Abscesses should be drained as soon as they can be located. The drainage should be as thorough as possible. It is often impossible to wash the abscess-cavity through a drainage-tube, because the lungs are very liable to be flooded by the injected fluid, since bronchi also usually communicate with it. A large proportion of cases, if opened by puncture or incision and well drained, will recover. Occasionally a fistula will be left. In one case, under my own observation, a fistula persisted for more than a year, but was finally obliterated. Recourse to the same surgical procedures affords almost the only hope of successfully treating gangrene of the lungs. Many cures have been effected by incision and drainage. To give a patient the best possible chance, resort should be had to this method of treatment.

In both diseases the individual's strength should be conserved as much as possible. Rest should be maintained, both to prevent the employment of strength unnecessarily and, at least in gangrene, to more perfectly prevent the spread of the gangrene to other parts of the lungs, which might be caused by constantly varying the body's position. The appetite should be stimulated by bitter tonics when it is deficient, and digestion aided if it is imperfect. Food, under all circumstances, should be given with regularity, and in amounts sufficient to maintain general strength. The kinds of food to be employed, and their amounts, must be varied according to the condition of the patient's appetite and powers of digestion.

CHAPTER XIII.

PULMONARY TUBERCULOSIS.

Definition.—Pulmonary tuberculosis, or phthisis, is a specific inflammation of the bronchi and lungs. It is excited by the bacillus tuberculosis. The specific inflammation is always associated with simple inflammation, and usually with suppurative inflammation. It is a wasting disease, and commonly a chronic one.

Anatomy.—The anatomical changes which are observed in the lungs of those affected with tubercular disease vary greatly. They may be extensive, or slight, catarrhal, croupous, or interstitial inflammations. The lung is at first consolidated, and later excavated. While the anatomical lesions are so various, there are certain ones always present and characteristic; they are the tubercle-nodules. Miliary tubercles are usually present. Infiltrating tubercular tissue may also exist. The characteristics of the miliary tubercle are the formation by cells of a globular mass the size of very small shot. This body is composed of large numbers of small round-cells. Near its centre are larger, oval, epithelioid, and giant cells. To some extent within the cells, and more abundantly between them, tubercle bacilli can be seen. No new blood-vessels are formed in these nodules. When—and sometimes before—it attains its minute growth the cells at its centre lose their vitality, degenerate, and become a shapeless mass of dry fat-granules. This degeneration and desiccation constitute caseation. At first the miliary tubercles are gray and gelatinous, but soon become yellowish.

These minute masses may coalesce and form larger ones, which are sometimes called tubercle-nodules. Irregularly shaped or disposed lines of caseous material are also often observable. They may be made by the coalescence of miliary tubercles, but they may also be made by the degeneration of tissue that is infected by the tubercular poison. It is probable that some substance formed by the bacilli so affects the tissue-cells that they degenerate. The absence of capillaries, because of the non-formation of them in the inflammatory tissue and the frequent obliteration of them in old tissue, leads to the drying or caseation of the degenerated mass. The tendency to degenerate, though originating usually in a miliary tubercle, spreads from them, when they are completely involved, to the surrounding structures, providing the bacilli continue growing actively.

Tubercles are the foci of extensive simple inflammation, or they render chronic what was at first a simple inflammation. For example, around a miliary tubercle there may arise bronchitis, catarrhal or croupous pneumonia, and where tuberculosis infects the lungs, because of an existing bronchitis, it may make the latter chronic. Infection most frequently occurs first in the small bronchi, where a miliary tubercle is formed. It excites a catarrhal inflammation of the mucous membrane, as well as inflammation of the submucosa and deeper tissues of the bronchi. This may lead to several different results :—

1. Atelectasis may be produced if the inflammation and catarrhal desquamation produce obstruction to the minute bronchi. A patch of consolidation, lobular in size, will thus be formed. Soon, in the wall of some of the collapsed alveoli, miliary tubercles will develop, and

excite more extensive round-celled infiltration into the alveoli and interstitial tissue. In some of the collapsed air-cells catarrhal inflammation will be excited, and they will be filled with epithelial cells. Thus a tubercle-nodule is formed.

2. Instead of this course the localized tubercular capillary bronchitis may lead to peribronchitis and catarrhal pneumonia. The solidified tissue in this case, also, is lobular in size. It resembles in all ways the lesion of catarrhal pneumonia, except that it is infected by tubercle poison, and miliary tubercles may be formed in it, or it becomes extensively caseous under the influence of the bacilli.

3. Croupous inflammation of the lung may be excited. Oftenest the fibrinous consolidation is very limited in extent, but it may be extensive enough to involve an entire lobe.

4. To some extent in all cases, but especially in the most chronic cases, do the interstitial tissues become inflamed. Such inflammation produces broad bands of fibrous tissue. Tubercle-nodules and lung-cavities are frequently encapsuled by them. Cirrhotic inflammation is usually protective, because it tends to encapsule, and thus to limit the spread of the tuberculous infection. It helps to contract and to obliterate cavities. It produces the contraction of the lungs which is characteristic of chronic phthisis. In the fibrous tissue tubercle bacilli are very rare. It may almost be said that they do not exist in it. Apparently it is a barrier to their dissemination through the lungs. New connective tissue may encapsule a caseous nodule of any size and isolate it. All the cells containing bacilli finally degenerate; the bacilli die and disappear, and the caseous mass is thus rendered inert. It will then usually calcify, and may

remain in the lung indefinitely and harmlessly. Such protective capsules are not perfectly developed except in the chronic cases.

5. Ulceration of the bronchi may result from their primary infection. Caseation in a bronchial tubercle will spread through it until the submucosa is involved and the epithelium cast off. The cheesy matter will crumble off into the bronchus, and a loss of substance will be rapidly caused. Suppurative inflammation then sets in, and the bronchial wall is quickly eroded. A minute cavity is thus formed. A large one may be produced rapidly by the coincident destruction of tissue by caseation and suppuration.

6. Pleurisy is always excited when inflammation occurs immediately beneath the pleura. The latter is thickened. Adhesions between the pleural surfaces always exist when tubercular disease is widely diffused in the lungs, and often so extensively that the pleural sac is obliterated. The pleurisy which accompanies phthisis is usually dry. In a moderate proportion of cases it is serous, and rarely it is purulent. Miliary tubercles may form in the thickened pleura, but do not uniformly. Serous effusion occasionally occurs into the pleural cavity or into a part of it that has been divided off by preceding dry pleurisy.

Cavities are formed in two ways in phthisis: (1) by softening and excavation of tissues consolidated by catarrhal or croupous pneumonia; (2) by the dilatation of bronchi and their erosion through suppuration. The pathology of bronchiectatic cavities I need not repeat. (See page 57.) The influence of tubercle bacilli, or the chemical products of their life, is to cause fatty degeneration and caseation. This tendency is increased by the non-vascular character of tuberculous inflammation.

Softening and suppuration, though exceedingly common in caseous nodules in the lungs, is not as common in similar formations in other organs. It is undoubtedly true that softening and suppurative inflammation of consolidated and caseous portions of the lungs is often due to a superimposed infection by pyogenic agents. The latter is, then, the cause of liquefaction of the nodule. Koch's experiments with tuberculin show that chemicals the products of the growth of the bacillus tuberculosis may also cause suppuration. The softening usually takes place first in the centre of the mass. This is especially true if originally a bronchiole passed through its centre. The softening sometimes begins about the margins of a caseous nodule. At first the puriform fluid is odorless, thin, and contains few pus-cells, but large amounts of granular matter. Such pus is doubtless the result of tubercular infection only. Later, and especially after the cavity has opened into a bronchus, and it has become infected by the commoner pyogenic organisms, its contents are characteristic pus which is often more or less fetid. Minute cavities thus formed soon, by their growth, open into a bronchus and empty in part or wholly. They grow, as do other abscesses, by the degeneration and desquamation of the granulation cells that compose their wall. Bronchiectatic and other cavities in tuberculous lungs enlarge more rapidly because of the caseation which takes place here and there in their walls under the influence of the tubercle bacilli. For granular, cheesy matter, when laid bare, will rapidly crumble into a cavity and may produce a considerable loss of substance. After communication has been established with a bronchus, the air-pressure within the cavity is an important factor in enlarging it, for its walls are not firm, as a rule, and may be stretched.

Cavities frequently rupture into one another. Small cavities are usually irregular in shape; large ones are more frequently smooth within. But both vary greatly in these respects. Cavities almost invariably form first in the upper lobes; they may be numerous or there may be but one. In many instances numerous caseous nodules are observable between the cavities and the surface of the lung. As the excavations extend near to the pleura it is inflamed, and firm adhesions usually form between the pleural surfaces over cavities. A cavity may occupy the whole of one lobe, and be nearly as large as an infant's head. Bands of tissue often pass across them. These are usually parts of the interlobular septa, and they may include arteries of considerable size. The latter are usually obstructed by clots, but may become aneurismal and by rupture cause violent or fatal hæmorrhage. About all old cavities a fibrous envelope forms; if the cavity is perfectly drained this envelope may cause its contraction and even obliteration, provided the inner granulating surfaces of its walls can be brought in contact long enough for adhesion to be produced. Small cavities whose outlet becomes obstructed are rarely obliterated by the absorption of their fluid contents, by the contraction of their walls, and by the caseation or calcification of their solid contents. Unfortunately, this is not the usual course of pulmonary cavities; they must be expected to enlarge. The contraction of a cavity necessitates a compensatory dilatation or displacement of neighboring lung-tissue. Therefore the lung is often drawn upward from this cause, and if pleuritic adhesions are extensive other organs may be displaced. The contents of the cavities are pus, granular and oily matter. In all that are rapidly enlarging bits of the elastic frame-work of the lung can be found.

Tubercle bacilli and various micrococci, pus-forming and non-pathognomonic, are discoverable in them.

The capillaries in tubercularly-inflamed tissues become occluded, and new vessels do not form. The calibre of the larger arteries is sometimes diminished or obliterated by endarteritis. Occasionally small patches of hyaline degeneration can be observed in phthisical lungs. This change is usually limited to the fibrous tissues or the blood-vessels.

The various lesions that have been described are combined in many ways. Oftenest caseous nodules of catarrhal pneumonia are seen scattered through the lungs. Usually cavities are also observable at the apex. If the given case has been a chronic one, much connective tissue will be found about the lesions at the apex, while in the lower lobe more recent nodules of catarrhal pneumonia will be observed. Or, if life has ended from an acute exacerbation of disease, one lobe may be found consolidated by croupous pneumonia. Caseous nodules, or more diffuse masses of caseous material, will also be seen in it.

The tubercle bacillus, and, therefore, the cause of the characteristic lesions, is disseminated through the lungs by three channels: by the blood, by the lymphatics, and by the bronchi. In most cases dissemination occurs by both the last-named channels, and often by all three. In miliary tuberculosis the lesion's cause is always spread by the blood-vessels. In many chronic cases a few miliary tubercles will be found in the intestines. They are probably caused by infection from sputa that has been swallowed. More rarely other organs will contain them, which must have been infected through the blood. Infectious material is usually not carried far by the lymph. It may be conveyed from one part of a lobe to another part, and especially toward the hilus. The bronchial glands thus become involved. But it is not probable that the bacilli are transmitted by these channels from one lobe to another, and certainly not from one lung to the other. The bronchial tubes are most frequently the channels of dissemination. The sputa is its carrier. Sputa is not always expelled when it is moved in the bronchi, but is often drawn back, and even carried into the lung-tissue, by the air-currents. J. K. Fowler has recently demonstrated quite conclusively the usual path of infection by the bronchi. Oftenest the primary trouble is at one apex. The difference in the susceptibility of the lungs is slight: the left is probably first affected a little the oftenest. The lesion does not develop absolutely at the apex, but an inch or thereabouts below it; or else, opposite a point on the exterior of the chest, just below the outer third of the clavicle. The lesion is also usually nearer the posterior surface of the lung than the anterior. Before the opposite lung is involved the top of the lower lobe on the side first infected becomes the seat of a lesion. The opposite lung is then attacked, and generally its different parts are involved in the same order. The middle lobe is last involved, and often escapes entirely.

The primary lesions near the apices of the upper and lower lobes increase in size and extend particularly downward. New nodules also form close by, and, growing, finally unite with the first ones. In this way the solid mass increases. It has often a rudely-triangular shape, the triangle's base being the primary nodule, the apex being downward and the anterior border parallel, and usually coinciding with, the anterior surface of the lung in the upper lobe and the interlobular septum in the lower. Beneath these largest solid masses smaller discrete ones can be found.

The way in which the diverse lesions of phthisis are produced is not perfectly clear. Undoubtedly the bacillus tuberculosis causes, by its presence or by the products of its vitality, miliary tubercles and diffuse tubercular tissue. The catarrhal and peribronchial pneumonias are the results of the extension of inflammation which accompanies or surrounds what is strictly tubercular. The interstitial inflammation is undoubtedly chiefly conservative. The presence of tubercle bacilli, or, more probably, of the chemical products made by them, leads to caseation. It is not common for tubercular lesions elsewhere than in the lungs to suppurate; when they do, other microbes than the tubercular are found in the pus and inflamed tissue. It is therefore not clear that suppuration of tubercularly-inflamed tissue is often due to the tubercular infection only. The tubercular inflammation excites general symptoms as well as local ones. The former are very like those of a chronic septicæmia. This is especially true after suppuration, when usually a genuine septicæmia exists.

Miliary tuberculosis of the lungs develops when the blood is infected. The miliary tubercles will then develop simultaneously in most of the organs and tissues of the body. The disease is a general one, and not strictly one of the lungs. In such cases the tubercles are uniformly scattered through the lungs. They may be so numerous as to fill a large part of the lung-tissue, or they may not be at all numerous. They may coalesce to form small nodules.

The lesions of so-called acute pulmonary phthisis do not differ from those of the chronic form, except in the rapidity with which they develop and spread. Fibrous tissue is rarely developed, or, at least, to any considerable extent. Miliary tubercles may not be present. Often one entire lobe is consolidated and extensively caseated or excavated. The lesions may be catarrhal or croupous, or both. They caseate with rapidity, and cavities form in them as quickly. Gangrene not unfrequently supervenes.

Symptoms.—In *chronic tubercular* disease of the lungs three stages are recognized clinically: a stage of incipient tuberculosis; a stage of consolidation; a stage of softening and excavation. The first is anatomically characterized by a localized bronchitis, usually in one or both apices, and by consolidation too small in amount to recognize clinically; the second by clearly recognizable consolidation; the third by the formation of cavities.

In the first stage the physician may be consulted either for a gradual loss of flesh and strength or for a persistent cough. Frequently, an acute bronchitis, which becomes chronic, though mild, is the origin of the disease. An insidious loss of strength, which causes lassitude and loss of flesh and color; are characteristic. The face grows gray or sallow, though the lips remain red. The patient tires quickly. His respirations are short and quick on slight exertion, and even when at rest the movements are shallower than is normal or they are unequal on the two sides. There is usually a slight, constant, hacking cough; the coughing is now and then aggravated by a fresh cold. Often the appetite and digestion remain normal, but symptoms of dyspepsia are not unusual. The pulse is quick, varying from 90 to 100 per minute. The temperature is slightly raised: when the bronchitis is severe it may be 102° or

103° F., but usually it is about 100° F. by eight o'clock in the evening, when it reaches its maximum. It is normal in the early morning hours. Not unfrequently a sense of chilliness is experienced about the middle of the forenoon, and, rarely, an actual rigor occurs. When the bronchitis is severe considerable sputa may be expectorated. It is in the early morning commonly muco-purulent, and later in the day frothy mucus; but the commoner, persistent, hacking cough is dry. In the sputa tubercle bacilli can be discovered if patiently sought for. Hæmoptysis may occur. Bleeding at this stage is small or moderate in amount; it does not occur in half the cases.

The physical signs are most characteristic in those who inherit a predisposition to tubercular disease. We discover this stage, usually, developing in boys and girls between the ages of 14 and 22. They are slender; their muscles are small and weak; their skin is thin and white; the chest is long, narrow, and thin; often the head droops forward or the shoulders bow; the anterior and upper surface of the chest is flat, and expands very moderately with inspiration. In those in whom the predisposition is acquired in adult life the shape of the chest is not characteristic. Respiration becomes shallow, and, usually, especially so upon one side. There is no abnormal fremitus. The apices are less resonant than is natural, but localized dullness is not discoverable. The respiratory sounds are not uniform over the chest: they may be exaggerated at both apices, but more frequently they are not uniform upon the two sides; for instance, in the left supra-clavicular region inspiration may be loud, in the infra-clavicular and supra-scapular regions it may be loud and often hitching. There is no expiratory sound. Over the lower part of the left lung the sounds may be clear and purely vesicular, but over the right apex the sounds may be low, and near the outer end of the clavicle they may be absent; a prolonged expiratory sound may be heard. Over the lower part of this lung the sounds will probably be low, vesicular. It must not be supposed that the respiratory sounds are always just as described in this illustration. The characteristic of them is rather a marked difference upon the two sides, and even in different parts of the same side, and especially an abnormality in the sounds at one or both apices.

In the second stage of the disease the bronchitis is more severe. Coughing occurs often, and is always a noticeable symptom, while in the first stage it is often so slight as to be overlooked by the patient. The expectorate is more constantly muco-purulent, and bacilli are more numerous. Often, in acute exacerbations, the usual symptoms of acute bronchitis will be present. Emaciation goes on more rapidly, and the patient is more languid. The appetite is often lessened and capricious. The pulse remains quick, is smaller and softer. The temperature follows the same course as in the earlier stage, but averages a degree higher by eight o'clock in the evening, and is usually a little subnormal in the earliest morning hours. Sweating at night may occur in the first stage, but less often than now. Respiration is shallower, and decidedly so upon one side of the thorax. It is more easily excited, and breathlessness on exertion is greater.

On inspection, the difference in the freedom of expansion of the two sides is evident. Often, especially in the chronic cases, the supra- and infra-clavicular regions upon one side will be more retracted than upon the other. Vocal fremitus is increased over these retracted areas. At these points there is greater relative dullness, but nowhere is there absolute dullness. Over these same areas the respiratory sounds are broncho-vesicular. Moist râles can occasionally be heard. These evidences of lung consolidation may not be confined to one apex, but may be elicited over areas of either lung, or different parts of each. They are discoverable, usually, at the apices, or posteriorly at the top of the lower lobes. Over other parts of the lungs the physical signs may be normal, or those of bronchitis only.

In the third stage of the disease the emaciation is often extreme. The cheeks are hollow. If the invalid attempts to sit up, the back quickly bows and the shoulders sag. Speech is frequently slow. There is little endurance, and sooner or later the patient is bedridden. The pulse grows smaller and remains soft and quick. The temperature varies much from day to day, but averages 102° F. at night, and is almost uniformly subnormal in the early morning. Night-sweats are often of daily occurrence. The cough is constant, but varies greatly from time to time, and in individuals as regards severity and frequency. It is accompanied by the expectoration of a muco-purulent or purulent sputa. If the cavities are enlarging by the disintegration of lung-tissue elastic fibres can be found in the expectorate. The appetite is variable, but is usually diminished or very capricious. Pain in the chest may be felt in any stage of the disease. Oftenest it is of pleuritic origin; more rarely it is neuralgic or rheumatic.

Inspection of the bare chest demonstrates the great emaciation which has occurred. The lungs do not expand equally, and respiration is much quickened. Palpation reveals increased vocal fremitus over areas of consolidation. Not unfrequently a bronchial fremitus can be felt. Resonance is lessened when there is consolidation, but is tympanitic or semi-tympanitic over superficial cavities. Over areas of consolidation the respiratory sounds are broncho-vesicular, or bronchial; over cavities they may be cavernous or metamorphosing; or, constant bubbling râles may be heard at one point, râles that do not move or disappear after a cough, or with changes of the patient's position. Often, however, we must rely upon the discovery of elastic fibres in the sputa in order to prove that excavation has begun or is progressing.

Toward the close of life the pulse grows more thread-like; the skin becomes moist and gradually cold. Respiration is very shallow and may be labored. Cyanosis is apparent. The mind may remain clear to the last, but oftenest consciousness gradually is lost as cyanosis deepens. During the last few days of life, or at least during the last few hours, coughing becomes infrequent or ceases, or if it occur is unaccompanied by expectoration. Mucus and œdema gradually obstruct the bronchi and lungs until respiration is impossible.

I have described the stages of chronic tubercular phthisis as though they followed each other rapidly, but they may not do so. Periods of quiescence or of partial recovery are the rule. They may occur between the various stages, or in their midst. For example, some pulmonary consolidation may be developed, and then apparent restoration to health may occur. After months or years an extension of consolidation may take place, or a cavity may form. It is these periods of quiescence that make the disease so eminently chronic. During them the physical signs of some consolidation or of a cavity will remain The tempeature may be normal in range, but its daily curve usually differs from a normal one, in that the maximum point occurs late in the afternoon instead of early.

It is important to know what are symptoms of improvement. One of the first that is noticeable is disappearance of temperature or prolonged remissions. The pulse becomes slower and often fuller. Flesh is gained. It is true, that rarely flesh may be gained while the fever persists. An individual's change in weight is so good a criterion of the course the disease is pursuing that it should be frequently observed. A gain in weight is significant, at least, of a diminished general tubercular intoxication. A sign of improvement still more important is an increase of respiratory capacity. It means that the lungs are filling more perfectly, and, usually, that the croupous and catarrhal exudates are at least in part being absorbed. This change does not occur, as a rule, except during remissions in the disease. Changes in the respiratory capacity are best measured by the spirometer. When very considerable increase takes place, measurements of the chest's girth at the end of deep inspiration will demonstrate it. The physical signs change if improvement occur in the condition of the lungs. It must be remembered that improvement in a consumptive's general condition often occurs without a diminution in the extent of diseased tissue in the lung, and is coincident only with a cessation in the activity of the disease. If areas of consolidation not only cease to increase, but also contract and become encapsuled, dullness will diminish, and all other physical signs of consolidation will be less noticeable, or will disappear. If a cavity cease to grow or it contracts, the signs of its existence will be less diffuse or evident. Elastic fibres will disappear from the sputa. The latter will become less purulent and less abundant.

The tubercle bacilli will diminish in number and may disappear. Improvement may go so far that a consumptive may be able to accomplish work equal to that of a man in perfect health, and still a cure may not be effected. To accomplish the latter, all tubercular poison that exists in the lungs must be destroyed, not simply rendered dormant to be rekindled later. The cough is one of the last symptoms to leave a case doing well. Its severity is never a criterion of the intensity of the disease.

There are several important complications, of more or less frequent occurrence. Indigestion is not uncommon. It is frequently the result of a catarrhal inflammation of the stomach. A disinclination for food is very common, but more than half the time it does not indicate indigestion; for if food is introduced into the stomach it does not ferment or cause distress; and, furthermore, experiments have demonstrated that the stomach secretes a normal gastric juice. Disorders of the stomach are suspected oftener than they occur.

Diarrhœa may occur at any stage of the disease. It commonly has two origins. It may be due to catarrhal inflammation of the intestine or to tubercular ulceration. The latter is a lesion of gravity, while the former rarely is, except when the individual, for other causes, is extremely weak, and cannot withstand even moderate additional exhaustion. It is often difficult to differentiate between catarrhal and tubercular inflammation of the intestines. If the tubercle bacillus is discoverable in the fæces tubercular inflammation may be confidently diagnosed, for it is very seldom that the bacilli are swallowed and voided from the intestines unchanged. Tubercular ulceration rarely occurs while the bowels remain constipated. In some cases symptoms of sudden peritonitis, peri- or para- typhlitis, intestinal perforation, hæmorrhage, or signs of internal bleeding first suggest the presence of latent ulcers.

The larynx may also be the seat of a complicating tuberculosis. In the earlier stages the laryngoscope will reveal swelling of the larynx, especially pale, nodular swelling. Later, ulcers can be seen. Hoarseness and even aphonia are common. Sharp, piercing pains are usual. Swallowing is often so distressing that food is refused, and all attempts at deglutition are avoided.

Hæmorrhages may occur at any stage of the disease. When slight, only streaks of blood are seen in the sputa; when moderate, a few mouthfuls of bright-red and usually frothy blood well up into the throat. In severe cases large quantities will be thus voided for hours or even days at a time. When large amounts are raised vomiting may be provoked, or the blood may simultaneously flow from the mouth and nose. Hæmorrhage from the lungs is accompanied by coughing. Hæmoptysis is distinguished from hæmatemesis by an accompanying cough with the former and vomiting with the latter, by a brighter redness of the blood from the lungs and blacker hue of that from the stomach, by the alkalinity of the former and acidity of the latter, by its absence from the stools of the former and presence in those of the latter, by the existence of preceding disease of the lungs in one case and of the stomach in the other. Hæmorrhage from the lungs is rare for any other cause than tubercular disease, and is, therefore, an important diagnostic symptom. It is seldom dangerous to life.

Pleurisy is so constant in its occurrence that it must be regarded as part of the disease rather than as a complication. Often it is chronic, and produces so little pain and symptoms so insignificant that it escapes attention. In a large number of cases it causes characteristic pain and other symptoms. Not unfrequently tuberculosis is the cause of pleurisy with effusion. In the beginnings of phthisis the effusion may fill an entire pleural cavity; in the later stages, after adhesions between the pleural surfaces have become extensive, effusions are usually circumscribed or pocketed. Pleuritic pains are often very distressing and in many cases recur frequently. They may be felt at any time during the course of the disease.

Renal lesions do not often form complications. Renal tuberculosis may develop. More frequently amyloid infiltration of the kidney occurs. The commonest hepatic complications are fatty degeneration and amyloid infiltration.

What is called quick consumption or acute phthisis may vary greatly in its course and mode of development, the common feature of all cases being the rapidity of the course. In many cases the symptoms are the same as those of chronic phthisis, but no periods of quiescence come in their course, and consolidation and excavation develop in quick succession. In many instances, during a few weeks preceding the actual outbreak of the disease, a slight cough and noticeable loss of flesh will attract the patient's attention. Suddenly a sharp pain will be felt in the side. The temperature will rise to 104° F., or thereabouts, and for a few days perhaps be continuous, but will soon grow irregular and approximate a hectic type. Emaciation will progress rapidly. The cough will be hard and painful at first. The expectorate may at first be mucous, later mucopurulent, and, when excavation progresses, purulent or gangrenous. Tubercle bacilli can be found in it. The appetite is diminished. The bowels may be constipated or irregular, but often, toward the close of life, they are loose. The pulse is soft from the first, grows small, and is constantly quick. Respiration at first is painful, on one side at least, and therefore expansion of the chest is lessened, and its movements are superficial. Almost from the first large areas—lobar ones often—are consolidated by croupous inflammation or by a combination of it with catarrhal inflammation. Therefore, percussion soon reveals dullness over these areas; auscultation, bronchial respiration; and palpation, increased fremitus. Usually coarse, moist râles are abundant from an early date. The disease runs too short a course for much contraction of the lungs to develop. In a few weeks after the consolidation excavation begins and progresses with rapidity. It is revealed by the usual physical signs, and by the presence of bits of lung-tissue in the sputa. In several instances I have seen gangrene supervene. The duration of these cases varies from six weeks to three months.

Diagnosis.—A diagnosis is made directly by the history of loss of flesh, by the existence of a small, soft, quick pulse, an irregular but persistent fever, the physical signs of a circumscribed bronchitis or pneumonia, the existence of tubercle bacilli in the sputa, and, in the stage of active excavation, of elastic fibres in it.

It can be differentiated from acute bronchitis by the persistence of fever, by the greater loss of flesh, and by the limitation or concentration of the physical signs at the apex of one or both lungs. In simple bronchitis the inflammation is quite uniformly diffused in both lungs. After consolidation and retraction have occurred, it cannot be confounded with bronchitis, although it might be with interstitial pneumonia and peribronchitis. The latter affections are not accompanied by the hectic fever and progressive emaciation which are inseparable from phthisis. In the stage of excavation elastic fibres are significant, although they may be found in the sputa that comes from simple abscess and gangrene of the lung. In the stage of softening it can hardly be confounded with any other disease, because of the history of its development, its chronicity, the chiefly apical location of its lesions, the simultaneous existence of areas of consolidation, retraction, and excavation. The discovery of tubercle bacilli in the sputa at any stage makes a diagnosis a positive one.

Causes.—So generally is it admitted that tubercular inflammation is due to the bacillus tuberculosis that I have not discussed the history of the growth of knowledge in regard to it. Belief that this bacillus is the cause of the disease is based upon the facts that (1) it is always found associated with its lesions, and (2), when isolated and inoculated into animals, it produces characteristic tubercular lesions. A few cases of accidental inoculation of man have borne the same results.

It is found that the bacillus does not produce the disease with equal readiness in all animals, and that all men are not equally susceptible to it. Therefore, there must be other predisposing causes or susceptible states of the system. A predisposition to the disease may be either inherited or acquired. It is very rare, if ever, that the bacillus is transmitted to the child while in the uterus; but tuberculous and cancerous or otherwise very feeble parents usually give birth to children who are peculiarly susceptible to the disease. Many infants are infected by the milk of tuberculous mothers or nurses, and usually become scrofulous, or develop intestinal tuberculosis.

There are several factors by means of which a susceptibility may be acquired. Usually several of these factors act together. Persons who breathe air that is confined in poorly-ventilated rooms, and especially air that is simultaneously breathed by many persons, are peculiarly apt to develop a susceptibility. They are liable to the malady both because their general vigor and ability to withstand disease is lessened by breathing such air, and because the air is especially apt to contain the bacilli.

Lack of exercise is a second factor that aids in developing a predisposition. If general exercise is wanting, vigorous health and powers of resistance cannot be maintained. If exercise, such as will insure frequent, deep breathing, is wanting, the lungs will be imperfectly expanded and air will remain in them long unchanged. If the lungs are thus imperfectly ventilated, it is possible for the bacilli to gain lodgment and to remain long enough to grow. As they grow with peculiar slowness, good pulmonary ventilation will greatly help to prevent infection.

Foods may be a source of infection. Cows' milk and beef sometimes contain tubercle bacilli, and when eaten may cause tuberculosis. This, however, rarely occurs, except in infancy, when raw, infected milk may be taken for weeks and months consecutively. Healthy digestion often, but not always, kills the bacilli. Cooking always will. There need be little fear of tuberculous food if it is not taken raw. A lack of nutritious food will cause a degree of general feebleness which makes one little resistant to any form of illness.

Other pulmonary diseases, especially chronic ones, predispose to tuberculosis of the lungs, by removing the natural guards of the respiratory passages against infection. Bronchitis often destroys the ciliated epithelium of the bronchi, whose function it is to keep the latter clean. Excoriations or superficial ulcerations make it comparatively easy for infectious germs to gain access to the deeper interstitial tissues, and to lodge in the air-passages long enough to multiply. We therefore find pulmonary tuberculosis often following other inflammatory affections of the lungs.

Diabetes is a general disease which, with peculiar frequency, is followed by or associated with tuberculosis. It certainly creates a susceptibility to the latter disease.

Both sexes are affected with equal frequency. Pulmonary tuberculosis may develop at any age, but it is least likely to in the earliest and latest years of life. Most frequently the earliest symptoms can be detected in the latest years of youth and earliest years of adult life.

It is a disease that is ubiquitous. It can be found in all climates. It is, however, not equally common in all. In general it may be said that in the most sparsely peopled regions of the world it is most infrequent. High altitudes and high latitudes are most exempt from it. The coldness of such localities leads to purity of the air. The rarefaction of the atmosphere in high altitudes also contributes to its purity, especially to its freedom from dust, because the latter is not easily suspended in thin air. The habits of the inhabitants of such regions necessitate vigorous exercise out of doors, and the stimulating qualities of the dry, cool air incite them to it. This insures deep and frequent breathing of pure air and the maintenance of a good circulation. The rarefied air of high altitudes necessitates deep breathing, involuntarily trains the muscles of respiration, and develops voluminous lungs.

Pulmonary tuberculosis is also somewhat less frequent upon dry, well-drained soils than upon low and poorly-drained ones. The disease is most abundant in temperate and warm climates, where the soil and air are damp and the temperature is changeable.

Pulmonary tuberculosis is a constant scourge and is more deadly than the epidemic diseases. On an average, 1 in 7 of all who die, the world over, succumbs to it. In certain localities the mortality is much greater, and in others less. In many, especially in old prisons, from 50 to 70 per cent. of the deaths are from this disease. Clean, fresh air is so important for its prevention that good ventilation of rooms and good ventilation of the lungs, maintained by exercise, have lowered high mortalities from it in the inhabitants of prisons, barracks, schools, and monasteries.

Prophylaxis.—Prophylaxis is extremely important. We can hope that, by a proper regulation of the life of the people, it may be possible to greatly diminish the frequency of the occurrence of pulmonary phthisis. This must be accomplished by preventing infection, by removing an inherited predisposition, and by preventing its acquisition. Tuberculous milk, whether it comes from a mother or from cows, should not be fed to infants. Tuberculous milk and meat are less apt to infect adults, for they rarely take much of either in an uncooked state.

As infection takes place almost exclusively through the respiratory passages by means of contaminated air, it is self-evident that pure air only should be breathed. To prevent contamination the bacilli should be destroyed as far as possible, and perfect ventilation of living-rooms and shops should be maintained. The air is contaminated by tubercular matter only when the latter is dried and forms a part of the atmosphere's dust. Such dust is almost exclusively formed by the drying of sputa. When it is remembered that sputa is cast upon the floor of buildings, upon the ground, and upon handkerchiefs or other articles of dress, and permitted to dry and be scattered by air-currents, the abundance of the contaminating material becomes evident. For the safety of others, tuberculous patients should be instructed to always expectorate into vessels filled with water, or upon handkerchiefs or other articles that can be boiled or burnt before they dry. Cuspidors should not be emptied upon the ground, but into sewers or upon a fire. They should often be thoroughly scalded.

If sleeping-rooms, living-rooms, factories, offices, and halls, where much of our life is spent, are well ventilated, the air will be constantly diluted and purified, so that the chances of infection will be greatly lessened. The fact that those whose occupations or modes of life necessitate their breathing a close and confined air, and especially one that many are simultaneously breathing, are more subject to the disease than others, establishes the need of fresh air for healthful living. Some years ago it was found that, among certain English soldiers, the mortality from consumption exceeded very greatly that among the towns-people about them. The old and poorly-ventilated barracks that they were then using were torn down and replaced by new ones, especially constructed to maintain ventilation as perfect as possible. The result was, that the mortality fell to as low a point as in the healthiest districts of England. A life out-of-doors—if possible, in the country—should be led by those predisposed to the disease.

The bad influence of a sedentary life upon those predisposed, and its influence in developing a predisposition, emphasizes the need of exercise as a means of prophylaxis. Exercise should be general to maintain a good degree of nutrition and a vigorous circulation. They must, also, often be especially adapted to develop the lungs and to increase the forcefulness of respiration. In those whose build predisposes them to consumption the lungs are unusually small in vertical diameter, but very long, and the heart is small. General exercise will strengthen and enlarge the heart. Pulmonary exercise—that is, voluntary, deep, full breathing—will enlarge the lungs, strengthen the respiratory muscles, and create a habit of deep breathing. Life at high altitudes, hill-climbing, and running especially lead to involuntary deep breathing. In young people the chest is mobile, and can be shaped and enlarged by persevering exercise. It is often necessary to correct other muscular weaknesses which produce deformities that hinder good respiration: such are round shoulders and stooped neck. They are due to weak shoulder- and back- muscles. For children and youth who are physically deficient special physical training is needful.

Daily baths or douches, and thorough rubbing after them, are useful in training the peripheral vessels to dilate promptly and restore warmth to the skin when it is suddenly chilled. If this habit can be acquired by the vessels the evil effects of sudden and violent atmospheric changes will be lessened.

The body should be constantly covered by woolen garments. They may be light or heavy, according to the season, but in changeable climates they should be worn throughout the year. It is best, also, that woolen night-garments should be worn. The advantage of woolen clothes is, that they maintain within themselves an atmosphere that is slowly influenced by external changes. They are porous, and do not keep upon the skin exhalations which should be carried off in order to maintain cleanliness. Certainly, colds are less frequently taken or aggravated by those who exercise such care in dressing.

The climate best adapted to those disposed to the disease will be indicated when the climates which are most suitable for the different stages and varieties of consumption are described. For those especially susceptible out-door employments should, by all means, be preferred. Close confinement to a desk or at trades, such as sewing, tailoring, and shoe-making, should especially be avoided; particularly should such individuals not work in poorly-ventilated rooms.

If the tonsils of a child are chronically enlarged and, by their mechanical interference with respiration, prevent the proper development of the chest, they should be removed. Respiratory affections, especially such as subacute or chronic bronchitis, should be cared for as quickly as possible, for they also make the individual affected more susceptible to infection.

Treatment—Hygiene.—We know of no specific for tuberculosis. Medicinal treatment is therefore symptomatic. Hygienic treatment is all important. Hygienic measures, as well as medicinal treatment, must be adapted to individual cases. Pure air is as essential to the consumptive as to those predisposed to the disease. The purest air is found in mid-ocean and upon mountain-tops. It is better in the country than in the city. At high altitudes—that is, at elevations of more than five thousand feet—advantage is derived not only from the purity of the air, but from its rarefaction and consequent influence upon the depth and frequency of respiration. High altitudes are especially indicated for young people who are predisposed to the disease, or have it in its incipiency, provided they do not have fever. It is indicated for fibroid phthisis, especially if it occur in young persons. It is also indicated for those suffering from any variety of chronic phthisis during a period of quiescence. But it is not so uniformly beneficial in those who are past middle life or very feeble. The rarefaction of the air causes involuntary, deep, and, at first, frequent respiration. Little by little the lungs expand, so that their capacity is increased. As this change takes place the breathing becomes slow, but remains unusually deep. The deep and strong respiratory movements that are thus constantly necessitated enforce a better ventilation of the lungs, a better oxygenation of the blood, and, therefore, more active tissue-change throughout the body, and a strengthening of the respiratory muscles. The increased capacity of the lungs is brought about by distension of the air-cells which, in lower altitudes, are only partly expanded and little used, and often by rendering other portions emphysematous. This helps to prevent infection by maintaining good ventilation of the lungs. Emphysema is usually produced about areas of consolidation. The stretching of the lung-tissue here and consequent stretching and narrowing of the capillaries prevent congestion. The dryness which is characteristic of high-altitude atmospheres causes the exhalation of unusually large quantities of water. This aids in bringing about an absorption of inflammatory exudates. The breathing of pure air lessens the tendency to infection by pyogenic organisms, and the liability to form abscesses. Dry, clean, cool mountain-air is peculiarly invigorating to the nervous system. It often stimulates the ambitionless and lethargic to take the all-needed exercise. It gives greater buoyancy of spirits, High altitudes are contra-indicated in acute catarrhal cases, or in other forms in which there is an acute exacerbation. Fever is a contra-indication, since it is usually aggravated by high altitudes. Great debility is also a contra-indication. If the lungs are so extensively involved in consolidation or excavation that respiration cannot be well maintained in rarefied airs, the high altitudes are likewise contra-indicated. A very nervous temperament contra-indicates them; for the stimulating air may cause sleeplessness, extreme nervousness, and even muscular pains.

Many places in the Rocky Mountains are high-altitude climates *par excellence.* In this vast region employment can be found by those who can take it; and very excellent locations for permanent residence exist there. Of health resorts Colorado Springs and Manitou are the most famous. The Alps in Switzerland are also famous for the cure of consumption, but they are not as good as the Rockies, since permanent residence in high altitudes cannot so well be maintained there, for in the spring, when the snow melts, the air becomes damp, and exercise is limited by the wet and slush. In the Rockies snow lies on the ground only a short time and disappears with rapidity at most of its health resorts. High-altitude residences are few in the Apalachian Mountains, and when they exist they are on mountain-peaks that are frequently cloud-capped, enveloped in mist, and exposed to harsh winds; but residences at moderate altitudes of two thousand or three thousand feet are numerous. They afford climates that are stimulating and air that is pure, while, for those who can take exercise, the hill-climbing will accomplish nearly as much toward expanding the lungs and dilating the chest as the rarefied air of high altitudes. They are better suited, too, for those whose lungs are extensively crippled and who are greatly enfeebled.

Sea-air, which can only be had to the fullest advantage at sea, on a vessel, is especially suited to the same classes of cases as high altitudes, and also to persons past middle life and to those greatly weakened. It is characterized by purity; therefore, suppuration from superimposed infection is not likely to occur. It is invigorating to digestion and nutrition, and its moisture and equability make it palliative to dry, harsh coughing. Involuntary expansion of the lungs, enlargement of the chest, and prompt absorption of inflammatory exudates must not be expected. Pulmonary exercises, when indicated, must be voluntarily taken. The greatest benefit has been derived from high-altitude and sea airs; but, if the best results are expected, cases must be carefully selected for each. Sea-climates, because of their sedative influence, are indicated for the very nervous, who are too greatly stimulated by dry, high-altitude air.

Long sea-voyages, as well as prolonged residence at high altitudes, is essential to produce the desired effects. At least six months, and, better still, a year or more, should be spent in these climates. A sea-voyage is contra-indicated for those who are very weak or in danger of rapid failure, since they cannot turn back when started. It is contra-indicated for those subject to prolonged sea-sickness. A time of year and a direction should be chosen that will promise good weather. Three successive summers can be had by starting from the northern temperate zone and sailing across the Equator to the southern part of America or Africa or Australia, so that the winter will be passed in southern seas, and a return voyage made so that home is reached the following spring or summer. Boats should be selected that are not overcrowded with passengers and that are well provisioned. Good and varied food is essential for an invalid. A voyage in a sailing-vessel is the best for an invalid, for such boats go more leisurely and are less crowded than steamers.

A modified sea-air can be had upon many islands and coasts, which is often very beneficial. But it is neither so pure nor so constantly invigorating, because more changeable. Islands often resorted to are the Bermudas and the West Indies. Shore resorts are numerous. Those of California and Florida are peculiarly favorable to many cases of consumption.

It is desirable to remove many consumptives who, although in the early stages of the disease, are feverish and ill adapted to the climates already mentioned from the changeable and inclement weather of our northern cities. Those who are suffering in the advanced stage of the disease, if it is not making rapid progress, are also benefited by escaping winter and spring weather at the North. For persons who are naturally lethargic and need a stimulating climate, the dry, mild, and equable air of Southern California is peculiarly favorable. Its distance often makes it inaccessible. Western and Southern Texas afford an excellent winter and spring climate for those who need a moderately dry and warm air, and, therefore, one moderately stimulating and genial. Those who, because of the nature of their case, cannot make their home either at high altitudes or on or by the sea, are often most benefited by transitory residence in southern States during the winter and early spring, and amid the pine-forests and moderate elevations of Virginia, Georgia, or New York in summer and autumn. Often those who cannot safely take advantage of a high-altitude climate, because of fever and the acuteness of their trouble, though in the first stage of the disease, can spend the winter with benefit near San Antonio, Texas. There they can live almost constantly out of doors and take the needed exercise, while the change will invigorate the appetite and usually cause a general improvement. Often their fever is gone before spring, and they can go with safety and the greatest advantage for a sojourn of a year or more to Colorado or New Mexico, where the mechanical effects of a rarefied air may be obtained.

Localities should always be chosen where there are few cloudy days, and where violent atmospheric changes are rare.

Often pure air must not only be sought by changes of climate, but it must be insured in the homes or places of business of consumptives. As little time should be spent in-doors as possible by those able to be out. Sleeping-rooms should be most thoroughly ventilated. Their windows should be kept open by day, and if it is necessary they may be moderately warmed before bed-time. Warm clothing and bedding should be at hand, but the temperature of the air need not be high. For those who are able to be about the house and out by day, it need not in winter be more than 45° or 50° F. if the air is dry, but must be much warmer if it is damp. For those who are quite weak it is best that the temperature of the room be kept uniform and at about 68° or 70° F. For those who cannot leave the house, but can leave their beds, it is best that two rooms be at their disposal, one to be used by night and the other by day. The one occupied by day should be sunny and cheerful; its windows should be opened wide by night, as the others are by day. At all times there should be permitted an egress of air that a constant change and freshness of the atmosphere may be maintained.

Suitably regulated exercise is quite as essential for the consumptive as for those predisposed to the disease. Exercise should be regularly taken, but should never be exhausting. By those who are feeble very little should be taken at a time, but in varied forms it may be taken often through the day. Short walks and rides are all they can bear. By those more vigorous and without fever field sports and games may be resorted to. These general exercises help to maintain muscular tone and vigor, and therefore a more perfect lymph circulation. The latter helps to eliminate much that might be detrimental if it accumulated in the system. This is probably the reason for the frequently-observed fact that, if accustomed exercise is not taken, night-sweats return and the appetite is lost. For those in the first half of life, and especially for those with fibroid phthisis, respiratory gymnastics are especially useful. These consist in enforced deep breathing. With the head erect and the shoulders back, as deep a breath should be taken as slowly as possible every two minutes while one is walking, and once in from half an hour to an hour while sitting in-doors. By this means the lungs are kept well expanded and the air in them most perfectly changed. The respiratory muscles are strengthened and trained to involuntarily maintain deep breathing. The chest can gradually be thus enlarged quite as much as by high-altitude life, provided only one will be sufficiently persevering. Another excellent but less-frequently available means of maintaining respiratory gymnastics is by the alternate inhalation of compressed air and exhalation into rarefied air, such as can be afforded by the pneumatic cabinet and similar contrivances. In this way the good effects of enforced deep breathing can be had, and the lungs can usually be more rapidly enlarged. Respiratory gymnastics, as well as general muscular exercises, must be gentle in the extreme when debility is great, or fever is continuously high, or cavities are large.

Hours for rest and sleep should be regularly provided. Many consumptives carry on business and give themselves insufficient rest. The clothing of the consumptive should be light, but warm. Too much clothing is frequently worn. It is burdensome, restricts the respiratory movements, and is not cleanly. Impervious garments, such as are made of leather, are not wholesome, for they make it impossible to dissipate the exhalations from the skin. Mufflers should not be worn over the mouth or nose, unless a high wind must be faced or a cold air excites a cough. In the latter case, they may be worn for a few minutes when the cold air is first struck, and until a tolerance of it is obtained.

For consumptives occupations should not be sedentary or necessitate confinement in-doors, nor should they cause mental strain or worry. The diet should be varied, but simple. As appetite is often poor, it should be tempted by a variety of food. If structural disease of the stomach exist, it may make necessary a still greater modification of the diet. It is usually necessary to crowd food upon consumptives, because of the lack of inclination for it. It is well to prescribe, in addition to such food as may be chosen at meal-times, a part or a full glass of good milk between meals and at bed-time. Milk is especially wholesome for those who like it. Butter, cream, and oils are also good, if easily digested. The fat-producers are the ingredients of diet that are especially needed by consumptives. Codliver-oil is peculiarly good, for it is easily digested. If it is taken, cream and other oils should not be too much urged upon a patient, lest his fat-digesting powers be overtasked. If indigestion exist oils cannot be taken. When they can they help rapidly to increase flesh, and with its increase other symptoms are ameliorated. The best preparations of codliver-oil are the clear, light-colored ones. They should be given to adults in as large doses as can be well digested. It is best to begin with a teaspoonful or less, and to gradually increase the dose. It is also best taken after eating. The taste may be made less objectionable if a little salt is taken before or after it, or if a bitter like gentian is added to it. The various emulsions are palatable, and by many readily taken. It is easy to administer the oil in elastic capsules containing from fifteen drops to a teaspoonful. If the taste of codliver-oil constantly return to a patient's mouth, or if it lessen the appetite for other wholesome food, it is not beneficial, and either the quantity taken must be lessened or it must be discontinued.

Malt-extracts are concentrated solutions of grape-sugar, with more or less of a diastatic ferment added. They have been recommended as a substitute for codliver-oil. This they are not. But they are good fat-producing foods, and as such may be used.

Forced feeding by a stomach-tube has been tried in this disease where there was great disinclination for food. It has been found that usually there is no disorder of digestion. Such feeding generally causes an increase of flesh and strength. It, however, does not occur if the destruction of the lungs is extensive, and if weakness is consequently very great.

Treatment, Medicinal.—Medicinal treatment must be palliative and symptomatic. The cough of tubercular phthisis is a chronic one. It may be influenced, as are coughs from other causes, by expectorants and anodynes; but as these remedies often nauseate, and usually diminish the appetite, they must be used with discretion. As a general rule, it may be said that they must not be used if they can be avoided. If coughing is severe and the expectorate is tight because of a fresh cold, the formula given on page 46, containing ammonium muriate, will be found serviceable. But such expectorant mixtures cannot be long used judiciously. In most cases an anodyne can be advantageously given at night to lessen the cough and insure sleep. The best anodynes are codeia, morphia, and chloral. The first of these is usually sufficient, and is the least objectionable. When the stomach is irritable, I have found a mixture of

Acidi carbolici, . . .		grm.	0.5 (♏vij) ;
Antipyrin, ...		grms.	10.0 (ʒiiss) ;
Tinct. gelsemii, . . .		"	15.0 (ʒiv) ;
Glycerinæ, ...		"	15.0 (ʒiv) ;
Tinct. opii camph., . .		"	30.0 (℥j) ;
Aquæ, . . .	q. s. ad	"	120.0 (℥iv) ;

to be serviceable. It allays the irritability of the stomach, and often lessens the cough in a surprising way. Teaspoonful doses may be given from every three to six hours. When the cough constantly needs mitigating, small doses of codeia may be given every three or four hours without disturbing the stomach, and with benefit. To loosen the expectorate when it is scant and tight the expectorants must be employed. If expectoration is abundant it can be lessened by terebinthines. Its purulent character may also be lessened by the same remedies. Beech-wood creasote has been lauded as especially curative for tubercular lesions of the lungs. I cannot think that, in therapeutic doses, it exerts a very powerful influence upon the bacilli in the lungs, for I have never seen the number of bacilli in the sputa lessened by it. It is certainly beneficial in rendering the sputa less purulent and less abundant. It does not, at the same time, make the sputa adhesive and difficult to raise, as does turpentine and its congeners. Creasote may be given in minim doses, and gradually increased to four or five times that amount, and the doses may be repeated every three to six hours. It is most agreeably administered in capsules, with gentian or pepsin or some other vehicle. A few times I have known it, in the larger doses, to cause some gastric burning and distress. More rarely, I have found the urine darkened, and exhaling the characteristic odor of the drug. In order to get most fully the best effects of creasote it must be given for weeks at a time. I have more frequently seen good results follow the persistent use of creasote than of any other drug. This good effect, I believe, is chiefly due to a diminution of the activity of suppuration.

When the larynx and trachea are much inflamed an inhalation of hot-water vapor, which has been impregnated with carbolic acid, or creasote, or turpentine, or pine-oil and paregoric, gives much comfort; it lessens the cough and the tracheal and laryngeal soreness. An inhalation of this kind can be best obtained from a flask partly filled with the medicated hot water and fitted with a cork, through which one glass tube passes to the bottom of the liquid and a second, shaped for a mouthpiece, into the vapor that fills the upper part of the flask. When inhalations are made the air is drawn in bubbles through the water, and is thus laden with moisture and is medicated. The inhaler should be used often if the inflammation is sharply acute. Steam-atomizers may be employed instead of the inhaler, but in my hands have seemed less efficient.

Codliver-oil and malt-extract, though more properly foods, often lessen cough, and may be used, at least, as adjuvants. Troublesome coughing can often be prevented by careful management. Many patients are most troubled by prolonged coughing at night when they retire and on awaking in the morning. The night-cough is due partly to the irritability of the nervous system from weariness, but more to a sudden change of position, and often of rooms and of clothing. The physical labor of disrobing and the stooping and bending which it necessitates are sufficient to provoke a spell of coughing, as can often be proven by having a patient assume the same posture and make the same movements at other times in the day. If a patient who is troubled with evening cough must climb stairs to a sleeping-room this should be done with great slowness, and a rest of fifteen to thirty minutes should be taken before clothing is removed. The room in which the patient undresses should be of the same temperature as that just left. One garment should be removed after another, slowly, and with frequent pauses for rest. It is best, usually, that cheerful conversation should be kept up, and the mind diverted from the expected siege of coughing. In many cases an attendant should help to remove the clothing, so that as little effort will be required of the patient as possible. The night-clothing should be warmed, so that it will not chill or shock the skin. In the process of uncovering the body, as little of it should be exposed to the air at one time as possible. The bed should be warmed. Often coughing can also be averted by not at once reclining in bed. The patient may at first sit in bed or lean against pillows, and then very gradually slip down into the bed and assume a recumbent posture. An hour or more should be occupied in leisurely getting to bed. The patient should, therefore, begin early. Very frequently, if those who retire at eight with a severe coughing spell will begin at five or half-past, and get finally settled by half-past six, they will avoid it. It is a mistake for consumptives to sit up late or to become too wearied. The morning cough is oftener difficult to stop, for it is usually caused by an accumulation of secretions in the air-passages or in cavities during sleep. If coughing occur occasionally during the night, but does not prevent the patient from falling asleep quickly again, it need not be checked, for it often prevents a wearisome and distressing spell of coughing in the morning. The morning cough can often be mitigated by taking a warm, nourishing drink on first awaking. A cup of warm cocoa is particularly grateful at this time. When coughing begins the patient should not sit up or get up, but should keep as quiet as possible. In this way coughing can be prevented from recurring with frequency. After the largest part of what is usually expectorated in the morning is raised the patient may begin to dress, but it should be done slowly, in a room whose atmosphere is genial. If the patient is feeble the hot, nourishing drink that has been recommended will be found especially beneficial if taken an hour or two, or even longer, before breakfast-time. Often, when the coughing spell begins at four or five in the morning, it will be mitigated by it, and an additional sleep will be obtained.

Anorexia is a very common and very troublesome symptom. A change of air and scene are often immediately beneficial. As the maintenance of strength by food is all-important, to counteract a disinclination for it becomes a necessity. By varying the diet, and by having all of its ingredients appetizingly prepared, the object may be accomplished. Oftener it is necessary to administer food in prescribed amounts and with the regularity of medicine. Milk, or some of its preparations, like kumyss, can thus be best given. It will often be more persistently taken if it is medicated. A bitter tonic may be mixed with it. A few years ago a decoction of mullein-leaves in milk was commended as one of the innumerable consumptive cures. The gain in weight and general improvement which followed its administration came from the amount of food that was thus forced upon the patient rather than from the drug. Bitter tonics, such as quinine, nux vomica, and gentian, are often prescribed, but in my hands have been of little avail. Such exercise as the patient can take and an outdoor life are especially serviceable in maintaining an appetite.

Vomiting oftenest is due to severe coughing, and will cease if the cough is lessened in severity by anodynes. More rarely it is due to gastritis, or other complicating disorders of the stomach. Generally, resorcin and bismuth will prevent it, or the carbolic-acid mixture described on page 154, from which the antipyrin may be omitted.

Diarrhœa and constipation frequently need treatment. The latter is amenable to the usual laxatives, such as aloes and cascara sagrada. The former may result from a catarrhal or tubercular inflammation of the intestines. They are both often persistent and recurring. If the diarrhœa is not severe the carbolic-acid mixture just mentioned may suffice to check it. In severer cases, and especially if intestinal ulceration exist, one of the following formulæ will be better ;—

1. ℞	Ol. gaultheriæ,	c.cm.	2 (ʒss).
	Ol. terebinth.,	"	10 (ʒiiss).
	Tinct. opii,	"	12 (ʒiij).
	Sacchar.,	"	30 (℥j).
	Acaciæ,	"	25 (ʒvj).
	Aquæ, q. s. ad	"	120 (℥iv).

Sig. : Make an emulsion and give in teaspoonful doses, diluted with water, every two to six hours.

2. ℞	Argent. nitrat.,	grm.	0.015 (gr. ¼).
	Pulv. opii,	"	0.015 (gr. ¼).
	Ext. gentian.,	"	0.12 (gr. ij).

Sig. : A pill, to be taken every four to six hours.

3. ℞	Plumb. acet.,	grm.	0.12–0.3 (gr. ii–v).
	Morphiæ,	"	0.008 (gr. ⅛).

Sig. : A pill, to be taken every four to six hours.

Numerous other astringents can be used, but none are more generally efficacious than those mentioned. Astringents and anodynes can sometimes be usefully given as enemata. Food must be administered with care, so that it will not irritate the bowels. It should be easily digested, and should not form bulky stools or contain irritants, such as seeds or fruit-stones.

Ferruginous preparations and the phosphates are indicated to relieve the anæmia which is almost constantly present in phthisis. They rarely meet the indication, unless they are given during periods of quiescence, when there is no fever and no active inflammation. Fresh air, sunshine, and good food are much more certain to stimulate the blood-creating tissues of the body.

The fever of phthisis is rarely treated. Antipyretics certainly only temporarily depress the temperature. The course of the fever is an intermitting one. In the milder and most chronic cases, when a rise of temperature is present at all, it is of short duration, and intervals of normal temperature are of some hours' duration. Whatever lessens the tubercular inflammation and suppuration will lessen or remove the fever.

Colliquative sweats are of frequent occurrence, and are often very persistent. They may be so mild as to be only a little annoying, or so profuse as to increase the sufferer's weakness. So long as they are not weakening, special treatment need not be resorted to. I say this because most drugs which are employed are not satisfactory, and those that are produce unpleasant side-effects. Slight and occasional sweating is sometimes due to a lack of air and exercise. It can often be lessened by salt, or alcohol, or vinegar, or other weak acid bath at bed-time. In those cases in which sweating occurs only in the early morning hours it can frequently be stopped by taking a drink of milk, or a little of some other food, in the middle of the night, or an hour or two before the sweating is most apt to occur. Ergot, strychnia, and digitalis, each alone or combined, will often do good for a time. They probably contract the peripheral vessels, thus lessening the blood-supply to the glands of the skin, and therefore their activity. A few drops of nitric acid or other strong mineral acid, given at bed-time or even several times daily, may be useful temporarily. The oxide of zinc is another remedy of value. Its mode of action is unknown. It may be given, in doses of 0.18 gramme (gr. iij), once to three times daily. The various preparations of belladonna, and especially atropia, are the most uniformly useful. Six tenths of a milligramme, a one-hundredth of a grain of atropia, administered at bed-time, will generally greatly lessen and often prevent the sweating. Larger doses may be needed, or it may have to be given two or three times during the night. To be efficient, it must generally be given in doses that cause dryness of the mouth, and at least slight dilatation of the pupil. These are effects that to many are more unpleasant than sweating. When atropia is efficacious in small doses it is probably due to its quieting influence upon the respiratory centre. When it has to be given often, and in full doses, it paralyzes the ends of the secreting nerves.

Hæmoptysis, if very slight, requires no treatment, but ergot may be given for a time to prevent its return. If at all copious, perfect quiet must be enjoined. Even coughing must often be suppressed by full doses of anodynes. Cold water to drink and ice to swallow help to prevent the bleeding. Frequently ice-bags may be placed upon the chest with advantage. Ergot is always useful, and can be given by the mouth or hypodermatically. Astringents, like the acetate of lead and tannic acid, are also given. Turpentine and the subsulphate of iron may likewise be administered by the stomach advantageously. Bleeding is rarely so copious as to endanger life, and, when it is, all these remedies may prove unavailing. When bleeding has ceased a recumbent posture should be kept for some hours, so that the obstructing clot will not be loosened by exercise. For the same reason the cough should be mitigated, and ergot should be given for some days.

Pleurisy is a frequent complication, and a painful one. It must be treated just as it would be under other circumstances. (See page 178.) Blisters are often used to check pleuritic inflammation. For this purpose they may be small,—an inch square or thereabouts. They also do good when there is fresh pneumonic consolidation. They then frequently hinder the extension of consolidation and mitigate the cough which accompanies it.

Since the discovery of the tubercle bacillus much has been hoped for from the employment of antiseptics. They have been administered by inhalation, by injection beneath the skin and into the lungs, by the mouth, and even by the rectum; but no positive cures have been effected by them. The best results have been obtained from antiseptic inhalations and intra-pulmonary injections. Unless it is desirable to modify fetid secretions or laryngeal and tracheal inflammations the inhalations accomplish little. It is true that, if a respirator is worn for hours at a time, all the air in the lungs may be more or less impregnated by the drug. The best antiseptics for use in this way are the volatile ones, like the terebinthines, oil of eucalyptus, thymol, creasote, and carbolic acid. Statistics do not show as good results for this treatment, when applied to all classes of cases, as for the constant breathing of clean, fresh air. Intra-pulmonary injections have sometimes done good, but the results obtained when they are used are so various that they have not won the confidence of the profession.

I need hardly speak of the numerous therapeutic fads which prove to be passing fashions and are useless as *cures.* To this class belong the rectal injections of sulphuretted hydrogen gas, recently tried so extensively, and the inhalations of very hot air, that have been tested still more recently. As yet no specific has been found for tubercular diseases.

A year ago Koch issued to medical men what is now commonly known as tuberculin. It has been very extensively and thoroughly tested as a cure for consumption. It is a glycerin extract of the products of the growth of tubercle bacilli in culture media. From it the bacilli and germs are perfectly removed, and only the chemical products of their growth remain. Tuberculin is a brownish, syrup-like fluid. A hypodermatic injection of 4 minims into a healthy adult will cause, in three or four hours, pains in the legs and arms, languor, inclination to cough, difficulty of breathing,—which is quite intense,—a protracted chill, and rise of temperature to 103.2° F. One-sixth of a minim usually produces slight pains in the limbs, transient fatigue, and sometimes a rise of one or two degrees of temperature. This is the smallest dose that commonly affects a healthy person. A consumptive, however, reacts moderately to one-tenth of this amount; therefore, treatment of this disease is usually begun with $\frac{1}{60}$ minim. Chill, fever, increased cough, and general aches are the symptoms which it commonly produces in the consumptive. After this dose has been repeated a few times, upon successive days, no symptoms are caused by it. The dose can then be doubled, and repeated until it produces no symptoms. Thus the quantity administered can be gradually increased until $\frac{1}{3}$ minim, or sometimes a little larger dose, is given, and no reaction is produced.

The mode of action of tuberculin is peculiar and extremely interesting. A year has passed since it began to be generally used. Though favorable results are still occasionally reported, its effects have generally been disappointing. My own trials of it have been uniformly discouraging. The drug is one of great virulence, and must be used with the utmost caution. My first trial of it was upon a young woman who had plainly slight apical contraction of the lungs, no cavities, only a hacking cough, no fever or night-sweats. I used about one-half the dose advised by Koch as a beginning one. No febrile reaction followed, but much soreness was produced in her chest. The same dose was administered on four different days, at intervals of from two to four days. There was no febrile reaction, but the soreness of the chest increased each time, and was so uncomfortable that I delayed increasing the dose or repeating its administration. Three days after the fifth injection a rise of temperature took place, and in a few hours she was confined to her bed with pleurisy. An extension of the areas of dullness took place rapidly. In three weeks a considerable cavity had formed at one apex, and at the end of ten weeks the patient died. I feel confident the tuberculin rekindled an old tubercular pleurisy, which in turn led to pneumonic infiltration of the lungs and rapid disintegration of them. I describe this case to illustrate the danger which even unusually small doses sometimes produce.

Tuberculin has the peculiar property of exciting active inflammation about tubercles. It does not do this—or accomplishes it very imperfectly—unless the tissue about the tubercles is somewhat vascular. Often very old and very young tubercles are not much affected by it. The inflammation which it excites sometimes causes encapsulement and oftener cellular degeneration. Koch says tuberculin can cause suppuration.

Chemical analysis shows that it contains albumoses, which constitute its active principles. By their separation, possibly, a less dangerous, but beneficial, agent may be discovered. The recent researches of Hunter and Koch give promise of this.

The utility of tuberculin must be looked upon as still unestablished. From the statistics thus far gathered it is evidently not of frequent advantage. It is a drug that must be administered with the greatest caution, and only to patients who can be closely watched. Each trial of it must be looked upon as an experiment.

Koch recommended it to aid in making a diagnosis.

He believed that only tuberculous patients would react to one-sixtieth of a minim. But it has been shown that tuberculous patients do not always react to it even when larger doses are given.

Prognosis.—The mortality from pulmonary consumption is very great. While its ratio to all deaths is estimated the world over to be one in seven, it falls as low as 1 per cent. in some localities, and rises to 60 and 70 per cent. in others. The frequency of the occurrence of the disease can be greatly lessened by improving the personal hygiene of the people, and still more by developing by physical exercises those children and youths who are prone to the disease because of defective growth.

The prognosis for those in whom the disease has begun its course must be guarded, but, unless great feebleness has resulted from the extent of the lesions or from their destruction of the lung, it need not be hopeless. Tubercular consolidation often is made harmless by encapsulement, degeneration, or calcification. Cavities may contract, and even be obliterated. Where statistics have been carefully collated in the autopsy-room, very numerous cases have been found in which tubercular lesions had cicatrized and become inert. In hospital cases, which, for the most part, come from the poorest people, because they neglect the beginning of illnesses, and in chronic ones continue to live unhygienically, the average duration of life after pulmonary consumption sets in is about two years. This is undoubtedly far from a correct average for those who can have early attention and can afford to care for themselves as they should. The average with them is from five to seven years. The statistics of Williams are the most extensive and reliable that have come under my observation.*

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* Pulmonary Consumption. By C. J. B. Williams and C. T. Williams.

Of his private cases, 36 per cent. lived from one to five years; about one-half of these died during the first three years. Sixty-nine per cent. lived from five to thirty years; about one-half of these died before the tenth year of the disease.

A prognosis cannot be based upon the number of bacilli in the sputa. If fever is constant, the chances of permanent recovery are not good. If there is a persistent loss of flesh they are not good. On the other hand, a gain of flesh is always a favorable sign. If suddenly a large part of a lung is consolidated by pneumonic inflammation, it is probable the course of the disease in that case will be short. If consolidation extend slowly and by small increments, and especially if long periods of quiescence occur in an individual case, a long course, and even ultimate recovery, is probable. The absence of fever and the possession of good muscular and mental vigor make it possible always to hold out hope of an ultimate recovery. Healing occurs oftener than is supposed. In nineteen thousand and fifty-three autopsies reported by various observers, ten hundred and thirty-two were found to afford evidence of healed tuberculosis. In other words, out of nineteen thousand persons, about one thousand, or about 4.7 per cent., had had consumption and recovered from it. It is also a noteworthy fact that death occurred in these cases of healed tuberculosis with great frequency from cancer (estimated by different observers at from 13 to 41 per cent.), heart disease (6 to 16 per cent.), and renal, bladder, and genital diseases (9 to 12 per cent.).

CHAPTER XIV.

NEOPLASMS OF THE LUNGS.

OF neoplasms only carcinoma and sarcoma are of importance or recognizable clinically. A diagnosis is difficult and often impossible, the disease being mistaken for some other chronic pulmonary affection. It may be secondary to new growths in any part of the body. Cancer of the breast is oftenest the source of the metastatic cancer of the lungs. It occurs most frequently in those who are advanced in years. The new-formed tissue may be circumscribed or infiltrating. Occasionally cancer occurs in miliary nodules. The pleura and the bronchial, cervical, and axillary glands are commonly affected. The cancer may be medullary, scirrhous, or epithelial.

In its earliest development a diagnosis cannot be made. Gradually increasing shortness of breath is felt, and often oppression across the chest. Sporadic and harsh coughing is usual. Sometimes pleuritic or neuralgic pains are distressing. The expectorate frequently has nothing significant in it, but becomes quite characteristic when it is reddish or blackish brown in color and gelatinous in consistence. Cancer-cells can sometimes be found in it. Occasionally the sputa is offensive. There is a gradual loss of flesh. The cancerous cachexia is developed, and, as in neoplasms elsewhere, loss of appetite, even disgust for food, and vomiting become prominent symptoms. Pressure upon the superior vena cava may cause œdema of the neck and side of the chest and arm, or the veins may be greatly distended. Pressure on the œsophagus may obstruct or prevent deglutition. Pressure upon the bronchial plexus or the involvement of the nerves in the cancer may cause intense neuralgia or paresis.

Physical signs are not pathognomonic. Irregular areas of dullness are usually found. If the bronchi are obstructed respiratory sounds and vocal fremitus will be wanting, or, if they are patent, the former will be bronchial and the latter exaggerated. Râles, and sometimes friction sounds, may be heard. Inequalities in the surface of the chest or prominences are significant.

The prognosis is unfavorable. Death generally occurs in from six months to two years, and is due oftenest to gradual loss of strength.

The treatment must be supporting and symptomatic. The same remedies are used as in other pulmonary affections for similar symptoms.

DISEASES OF THE PLEURA.

CHAPTER XV.

PLEURISY.

Anatomy.—An inflammation of the lining membrane of the pleural cavity is called pleurisy. First, a congestion of the pleural vessels occurs; the superficial cells become loosened and are cast off; the connective-tissue cells beneath swell and, it may be, multiply; the whole tissue is thickened by exuded lymph (which infiltrates it), by leucocytes, and, in spots, even by red blood-cells. An entire pleura may be inflamed; usually, however, only a part is affected. The inflammation generally varies in intensity in places. The pleura at one point may be thickly crowded with leucocytes and embryonic cells, at another only slightly swollen by the lymph. The serous exudate may be scant, may clot readily and deposit fibrin upon the pleural surface. It is then called *fibrinous*, and often *dry*, *pleurisy*. When it is purulent it is called *empyema;* when the serous exudate is so abundant that it accumulates as a mass in the pleural cavity it is called *pleurisy with effusion*, or *serous pleurisy*. Pleurisy may be *chronic* in character from its start. The pleura is then persistently, but moderately, congested; its connective-tissue and its superficial cells undergo considerable hyperplasia, and little or no exudation forms upon its surface. A permanent, and often very considerable, thickening of the pleura is thus produced. An acute pleurisy may persist and become chronic.

Whenever the raw visceral and costal pleural surfaces are held together they tend to adhere permanently, and thus to obliterate the pleural cavity or to divide it into compartments. They may unite, as it were, by first intention. Adhesions are often temporarily produced by a fibrinous exudate. A permanent band of tissue may supplant the fibrin if the latter become "organized." In this process the fibrin fills with embryonic cells, which, while they cause the fibrin to disappear, develop new and permanent connective tissue.

The results of fibrinous pleurisy are (1) resolution. The fibrinous exudate may be liquefied and absorbed, the swelling of the tissue may disappear, and a perfect restoration may take place. But more frequently (2) permanent thickening of the pleura or (3) adhesions are left. The lung is frequently crippled by these thickenings, since they prevent its full expansion. The superficial air-cells are often diminished in size or obliterated by an extension of the inflammation below the pleura into the lung's interstitial tissue, which becomes swollen, and often permanently thickened and indurated. Dry pleurisy may slightly contract or deform the thorax. The imperfect lung expansion, whose cause has just been explained, will lead to a depression of the overlying thoracic wall. Thickening of the costal pleura across intercostal spaces often prevents their expansion during respiration, and, therefore, the full distension of these portions of the thorax.

The result of a serous pleurisy, or pleurisy with effusion, may be resolution, with or without deformity. When serum partly or wholly fills a pleural cavity the lung must be correspondingly compressed. If the liquid remain in the cavity long enough for the visceral pleura to become permanently thickened so as to prevent or limit the lung's re-expansion and is then absorbed, the atmosphere's weight will cause the thorax to be pressed in to meet the collapsed or partly expanded lung. This is a common cause of permanent thoracic deformity in young persons, but it can produce only very moderate deformity in older persons, whose thorax has become rigid by more perfect ossification of its frame work. In them some liquid will remain if the lung cannot expand and the thorax cannot be compressed. Sometimes, even under these conditions, the liquid may be absorbed, and the cavity which it filled may be obliterated by an emphysema of the opposite lung, which will crowd the thoracic organs into the unexpanded side of the chest. Often an abundant serous exudate will be completely absorbed, the lung will perfectly expand, and no deformity will result. This takes place whenever the active inflammation is of short duration. A serous exudate may remain in the pleural cavity for a very long time. It may also become purulent.

More frequently an empyema is such from the beginning. Circumscribed empyema will rarely result (1) in absorption and caseation or calcification of the solid elements of the pus. Occasionally, therefore, we find after death, within the thorax, a plate of lime, which represents this process. The liquid of the pus was absorbed; its solids were dried and finally calcified. The pus may be (2) spontaneously drained by ulceration through the thoracic wall, which produces a fistulous channel. It may ulcerate into the pericardium, or through the diaphragm into the abdomen. These are fatal results. It also is (3) rarely drained spontaneously through the bronchi or stomach or intestines into which it ulcerates. If neither spontaneous nor surgical drainage is established, death results as from other large abscesses.

Exudates, both serous and purulent, cause displacement of the thoracic organs. A considerable accumulation of fluid in one pleural cavity will crowd the heart to the opposite side. Extra-pericardial adhesions may then produce its permanent displacement. The thoracic viscera are usually depressed. The lung always floats above the fluid, no matter what may be the position of the thorax. It is also more or less compressed. If the entire cavity is filled with fluid, it will be completely compressed, and may not occupy a space larger than one's hand. It is then non-vesicular throughout.

Causes.—Pleurisy may occur at any age, but is observed most frequently between the twentieth and fiftieth years. Males are somewhat oftener affected than females. Those who are in vigorous health are less susceptible to it than those who are feeble.

The malady is sometimes primary, but oftener secondary to some disease of the lungs or neighboring structures. The commonest cause of primary pleurisy is a wound of the thorax. A penetrating wound, or even a severe blow, will sometimes excite it. If the thoracic wall is deeply bruised, or for any cause inflamed, the pleura may be involved because of its continuity with it. A few clinicians deny that primary pleurisy can be produced except by a wound. But a majority believe that exposure simultaneously to damp air and to a sudden fall of temperature may, at least in feeble individuals, provoke it.

Pleurisy uniformly accompanies pneumonia unless the latter is entirely central, as it may very rarely be. It is almost as uniformly a complication of tubercular disease of the lungs. It always accompanies this disease unless the tubercular trouble undergoes resolution in its incipiency. Pleurisy may be one of the earliest phenomena of pulmonary tuberculosis, but more frequently it complicates the later stages. All other superficial inflammations of the lungs are accompanied by pleurisy. Peritonitis, abscess of the liver and spleen, and ulcer of the stomach or intestines may produce pleurisy by an extension of inflammation through the diaphragm. Pleurisy often complicates Bright's diseases. Pyæmia may be its cause. Miliary tubercles and cancerous nodules excite a surrounding pleurisy when they form in the pleura.

Symptoms.—Pleurisy is not always accompanied by symptoms. This is especially true of *chronic pleurisy.* Extensive pleuritic adhesions may be found in the thorax of phthisical patients in whom, before death, pleurisy was not diagnosed. Often persistent localized soreness about the chest, and especially at the upper part of the chest, in tuberculous patients, is indicative of chronic pleurisy, although there may be no characteristic physical signs or subjective symptoms.

Acute fibrinous or dry pleurisy usually begins with sharp, and often severe, pain in the side. The pain is localized, but the painful area may increase. It is aggravated by deep breathing, and sometimes even by restricted respiration. A dry cough is usual, but is suppressed as much as possible because it aggravates the pain. Respiration is quick. The thoracic movements are short, and upon the affected side less ample than on the other. Tenderness is experienced in all sharply-acute cases. It is limited, generally, to the intercostal spaces. A rigor, or a succession of slight chills, announces the beginning of acute attacks, and is followed by a fever. The latter may last only a few hours, or may persist for several days. It does not follow a typical course. It is rarely high, ranging, ordinarily, from 101° to 103° F. In subacute attacks these same symptoms are present, but are much less intense than in the frankly-acute ones. The physical signs of dry pleurisy are very important for making a positive diagnosis of the disease. Occasionally the palpating hand may feel a friction fremitus. Percussion usually reveals nothing abnormal. Sometimes, if the fibrinous exudate is unusually abundant, there may be a localized relative dullness. Auscultation demonstrates the characteristic friction sounds. They may be loud, but more frequently they are low. They are characteristically heard with each respiratory movement; sometimes, however, they are only audible with deep inspirations.

The physical signs of *pleurisy with effusion* are the same when the exudate is serous and when it is purulent. If, as is usually the case, the fluid enter a pleural cavity that has not been divided by pleural adhesions, the affected side will be observed to move much less freely than the opposite. It will appear fuller because the intercostal spaces do not show, and, therefore, the surface of the chest on that side seems smooth. It is also rounded as the normal angles are less acute. The apex-beat of the heart is often displaced. The displacement will be to the right if the left cavity is filled, and to the left if the opposite one is. Usually, and especially if the left pleural cavity contain the fluid, the apex-beat will be depressed because the diaphragm is. Vocal fremitus will be lessened, and generally is wanting. Percussion reveals an area of absolute dullness when the fluid has accumulated. Often it is bordered above by a semi-tympanitic space. This kind of resonance is due to the relaxed condition of the partly-compressed lung-tissue. If the fluid fill less than half of the pleural cavity, a change in the position of the body will cause the area of dullness to shift. It will be along the back if the patient is reclining, and will occupy the bottom of the thorax if he is sitting or standing. If much more than half the thorax is filled, it is difficult to detect the changes in the position of the surface of the liquid. The surface of the liquid is not perfectly horizontal. If the patient is sitting the line of demarcation between lung and fluid will be found to be curved. It is lowest at the spine, rises gradually to the axilla, and then falls a little to the sternum. This contour cannot be made out if the thorax is very full. The line of separation is also often difficult to locate, posteriorly, because the lung above the fluid is partly solidified, as it is not well filled if the patient is constantly in the recumbent posture. The curve which the surface of the intra-thoracic fluid produces is due to the displacement of it by the partly-expanded lung. Percussion better than inspection will demonstrate cardiac displacement, and, if the fluid is in the right pleural cavity, a depression of the liver.

The respiratory sounds are not transmitted through pleural effusions unless the lungs are solidified. Therefore, auscultation usually reveals an absence of respiratory sounds. Upon the unaffected side the sounds are exaggerated or puerile. If a pleural cavity is completely filled with fluid, bronchial respiration may be heard through it, especially toward its upper part. These sounds may be transmitted from the unaffected lung, but I believe are most frequently from the bronchi in a compressed and consolidated or carnified lung; for I have never heard the sounds except when the lung was in this condition, or the trachea and largest bronchi were imbedded in a mediastinal tumor, through which vibrations could be easily transmitted to the fluid. The reason that respiratory sounds are not ordinarily heard is not that fluid is not a good conductor of sound (for it is), but because the vesicular lung-tissue does not transmit the sound well to the fluid. The sounds are transmitted when the lung loses its vesicular character. It may, however, be as Garland urges, that such sounds are transmitted by the ribs and thoracic wall.

Friction sounds are often heard at the beginning of a pleurisy, and before the pleural surfaces are separated by the effusion. They may again be heard when the fluid is absorbed and the raw surfaces come together.

The physical signs of encysted effusions are the same, but the area of dullness which they cause does not change its position. This area can be mapped out, by percussion and by palpation, by noticing where the vocal fremitus disappears. If respiratory sounds are wanting within the bounds thus established we may feel confident that an effusion exists.

When a serous pleurisy begins often all the symptoms of a fibrinous pleurisy develop, but the pain ceases as the fluid accumulates in quantities sufficient to separate the inflamed surfaces. Respiration grows quicker and more difficult as the lung becomes compressed. If the fluid accumulate rapidly considerable dyspnœa may be felt. Soon the opposite lung expands, and will compensate for all moderate accumulations, providing the patient is at rest, but walking or any other form of exertion will quicken the respiration or cause dyspnœa. Cyanosis is not caused by pleuritic effusions, for the blood is well oxygenated by the healthy lung. Fever usually accompanies the outbreak of the inflammation, as it does that of fibrinous pleurisy, but it may last a day or two only; in other cases it persists for a week or longer. It does not follow any definite type. The heart at first is quickened by the fevered blood, and later its increased motion is maintained by the obstruction to respiration which the lung's compression causes, and often, in part, by its own displacement and consequent disadvantageous action. The pulse is usually not greatly lessened in size or firmness. If, as often happens, the fever is of short duration there may not be much loss of flesh or strength, but dyspnœa prevents exertion. A serous effusion may persist for many weeks. Usually it begins to be re-absorbed after two weeks or thereabouts, if resolution occur at all. Not unfrequently it becomes purulent.

The subjective symptoms of *empyema* are those of a serous pleurisy, with those superadded which are due to the absorption of purulent matter; therefore, the fever is hectic in type. Occasionally chills recur with each access of temperature, but more frequently the temperature follows a very irregular, intermittent course. Colliquative sweating is of nearly daily occurrence. There is progressive, and often rapid, muscular wasting. The pulse is soft, and of medium or small size. It is as quick as it is when a serous effusion exists. Death will usually result from the slow exhaustion which extensive and prolonged suppuration produces. It may result from some of the accidents which perforation may cause, as purulent pericarditis or peritonitis. The course of the disease covers a period of from one to four weeks, but, rarely, is more protracted.

Diagnosis.—The diagnosis of *dry pleurisy* can be directly made whenever friction sounds can be heard. When they are wanting, pleuritic pains must be differentiated from those of intercostal neuralgia and myalgia. Neuralgic pain is often intermittent, and is likely to occur when the breath is held and respiratory movements are not made. The three characteristic points of greatest tenderness are usually discoverable when the intercostal nerves are involved. Neither coughing nor fever are produced by it; it is true, however, that pleurisy may exist without either. Muscular pains are more shifting in character than pleuritic, and often are felt simultaneously about the arms or the opposite side. Palpation often makes it possible to locate them in a given muscle. A pleurisy sufficient to cause pain, and of a character not to cause friction sounds, will only occur when there is a chronic inflammatory lesion of the lung. If no such lesion is discoverable the pain can be diagnosed as not pleuritic.

The physical signs of an *effusion* into the pleural cavity are so definite that it can be diagnosed directly by the coincident (1) want of expansion of the affected side, (2) prominence of the intercostal spaces, (3) percussion flatness, (4) absence of vocal fremitus, and (5) almost uniformly of respiratory sounds. Pneumonia enlargements of the spleen and liver, and thoracic tumors have only dullness in common with pleuritic effusions, but in them the dullness is not absolute. After the existence of an effusion has been determined, it is necessary to ascertain whether the fluid is of pleuritic origin or dropsical (see page 194.), and whether it is serous or purulent. A hectic fever is suggestive of *empyema*, but is not positive proof. We can only decide positively after an experimental aspiration with a hypodermatic or aspirator needle.

Treatment.—The soreness which is often felt in chronic pleurisy, and the momentary but sharp pleuritic stitches which accompany mild acute pleurisy, are usually relieved by counter-irritants, A mustard-plaster may suffice, but a small fly-blister is surer. The latter need not be large. One an inch square is sufficient. In sharply-acute cases a larger blister is more effective, and should be followed by fomentations. Instead of applying counter-irritants the affected side of the chest may be "strapped." That is, strips of adhesive plaster may be so laid on that they will prevent or limit the motion of the ribs, and thus check the rubbing of the raw pleural surfaces against one another. Very great relief is often afforded by this procedure. The strips should be long, and should be applied at right angles to the ribs, so that they will bind them together and keep the intercostal spaces as small as possible.

Opiates must be used whenever the pains are very severe. Often drugs are contra-indicated, as they tend to diminish the appetite, or even to cause nausea. Frequently a small blister will be found to produce more permanent relief than an opiate. Morphine and codeia are the preparations oftenest employed. Just as in pneumonia calomel is frequently used (see page 101) because it seems to modify the exudate and prevent its being so fibrinous, and as it seems to promote the absorption of such exudates, it is used in fibrinous pleurisy. It may be given, at the beginning of the attack, in two or three doses of 1 grain each, or, perhaps more advantageously, in small doses of ¼ grain each, which can be administered for two or three, and sometimes four or five, days. Even mild salivation should be avoided. Purgation is usually not caused, as opiates must generally be simultaneously given.

Antipyretics are of little use unless the high temperature causes delirium, as it often does in children. Antipyrin or acetanilid will be found to afford relief to this symptom by depressing the temperature.

It is desirable that the bowels should be kept regular. At the beginning of the attack moderate depletion, by provoking a few watery movements from the bowels, does good. This can be provoked by administering two or three powders, at intervals of two hours, of calomel and bicarbonate of soda, containing 5 grains of each ingredient, or by giving the liquid citrate of magnesia, or some similar preparation. While there is fever the diet should be simple. If there is no fever the regimen of health may be followed.

The first or painful stage of pleurisy with effusion must be treated the same as fibrinous pleurisy. As soon as fluid is found to be accumulating the patient should be placed upon a dry diet, and liquids should be withheld as much as possible. A serous effusion can often be checked in this way, and its re-absorption hastened.

There are a number of drugs commonly employed to promote re-absorption of an exudate, but with doubtful utility. These are, especially, iodine painted upon the surface of the thorax; sodium chloride and potassium or sodium iodide administered internally. Common salt is occasionally given,—in as large amounts and as frequently as the patient can bear it. It is believed that if the blood can be made strongly saline a demand on the part of the system will be created for water, which will be satisfied by the absorption of the exudate if water is not drunk. The good effect of the iodides probably results from their increasing diuresis. They are not, however, as efficient diuretics as the acetate of potash or ammonia. Digitalis is usually combined with these in a diuretic mixture, but its employment must be governed by the rapidity of the pulse. If it is slow it may provoke vomiting, by making the heart too slow or irregular. Diuretics have almost invariably disappointed me when I have relied upon them alone to promote absorption of the exudate, but they are useful adjuvants to other methods of depletion. Saline cathartics may be used with them advantageously, but should not be pushed to that extent that they produce much weakness. Diaphoretics may also be used with one or the other of these methods of depletion. Sweating provoked by dry heat (see page 270.) is usually the best. Pilocarpine may be used, but it causes an enfeeblement of the circulation,—which is contra-indicated in some cases,—and often very uncomfortable salivation. Depleting agents cannot be successfully used if the patient is very feeble, and they often prove useless, even when employed as thoroughly as is possible. Aspiration affords a means of withdrawing the fluid promptly and surely, and, if properly done, without danger. If the amount of the effusion is large it should be preferred to any of the methods that are employed to promote absorption. The indications for aspiration are usually said to be: (1) if the effusion cause marked displacement of the heart; (2) if the fluid remain without change or increases in amount during three or four weeks. I have never felt justified in waiting three or four weeks before aspirating. If there is much fever, especially if it is hectic in character, I believe that one should not delay more than ten days before positively determining whether the fluid is serous or purulent. If aspiration is practiced, in order to establish a diagnosis some of the fiuid may as well be withdrawn while the needle is in place. If there is no fever it is not expedient to wait more than ten days before aspirating if the fluid does not in that time begin to diminish in amount; for the longer the lung is allowed to be compressed, the more danger is there that a thickening of its pleura or interstitial tissue will prevent its re-expansion.

If aspiration is to be practiced, the thorax can be best punctured in the lower part of the axillary space. The patient may sit or recline while the fluid is being withdrawn. If he is recumbent he should lie partly upon the affected side, but with it overhanging the edge of the bed. The fluid may be withdrawn until coughing is provoked, or decided distress in the side is produced from the dilatation of the compressed lung. It is not necessary to withdraw all the fluid within the pleura, for if its quantity is lessened an absorption of the rest will, as a rule, rapidly follow. Unfortunately, it will occasionally re-accumulate. Aspiration may be repeated as often as is necessary. It is only dangerous if the needles are not aseptic, or if a piece of emphysematous lung is accidentally punctured, for the former mischance may produce purulent inflammation and the latter pneumothorax. After aspiration, a physical examination will demonstrate that the area of dullness is lessened, and that respiratory sounds can be heard over a greater portion of the chest. In the rare cases in which reabsorption cannot be effected excision of the ribs may be resorted to; but it should be avoided unless every other resource fails after patient and persevering trial.

If pus is found in the pleural cavity it should be drained therefrom at once. A cessation of suppuration within the pleural cavity is effected by the permanent union of the visceral and costal pleura, which results in the obliteration of the cavity. This can only be accomplished while the lung is able to expand and fill the cavity after the pus is withdrawn. It is extremely important that an empyema should be drained as early as possible before the lung's expansibility is lessened by adhesions or a thickened pleura. Drainage is best established by making an incision through an intercostal space,—which is as low as will admit one to the pleural cavity,—and by inserting through it a large and closely fitting drainage-tube. A tube with a flange, which will prevent its slipping into the thorax, is to be preferred. If it is not at hand, this accident must otherwise be guarded against. If the operation is performed early and while the lung's expansibility is good a single drainage-tube will suffice, providing it is managed so that the lung is kept inflated. This can be accomplished by opening the drainage-tube only during inspiration, when the pus will be forced out by the dilating lung. During expiration the tube must be closely compressed. By thus pumping the cavity it can be thoroughly emptied. After it has been emptied in this way the tube may be temporarily corked. The procedure must be repeated whenever an ounce or two of fluid accumulates. This may, at first, necessitate its frequent repetition, especially if the pyogenic surface is extensive. But, usually, in a few days a part of the cavity will be obliterated, and it will have to be drained less and less frequently as less pus is formed. The pyogenic cavity can be washed with antiseptic solutions, and they can be aspirated out or pumped out in the same way that the pus is. This very simple method for preventing the access of air to the pleural cavity, and consequent compression of the lung, was first devised by Prof. Edmund Andrews, of Chicago. Others have contrived more elaborate appliances, containing a valve in the drainage-tube, that makes constant drainage possible. I have seen Andrews's method repeatedly employed, and with results that could not be bettered. A more usual method is to insert, at different points, into the pleural cavity several drainage-tubes, letting the air enter freely, and trusting to frequent washing and a depression of the thorax and distension of the opposite lung to obliterate the cavity. This is not so uniformly successful as the other method in the cases for which it is adapted. If, however, the lung cannot fully expand several orifices for drainage are usually needed, and should be so placed that they will make it possible to thoroughly drain and wash the cavity.

Often it is impossible to obliterate completely a large cavity, because the lungs cannot sufficiently expand or the thoracic wall sufficiently contract. In such cases resection of a part of the ribs may be resorted to, in order to bring together the pleural surfaces and make possible their adhesion.

Resection is a grave operation, and at the best entails much discomfort upon those operated on. I believe it should be resorted to only after other methods have been most faithfully tried and found unsuccessful. Occasionally a fistulous opening will persist in the side for months, from which a few drachms of pus will daily flow. The patient will regain flesh and strength in spite of this, and be able to do varied and even laborious work. Twice in just such cases I have seen resection tried and followed by a fatal result within two weeks. The prospect of months, and perhaps of years, of comparative health was good before the operation. I have seen in other similar cases such fistulæ and discharges persist for a year and more, and ultimately perfect recovery occur. If the pyogenic cavity cannot be easily closed, though drainage restore the patient to a condition of moderate strength and entire freedom from fever, I advise trying the inhalation of compressed air, or a residence at a high altitude, or enforced deep breathing and postural breathing, which will distend the lungs to the greatest extent possible.

Perfect drainage and antiseptic washings of an empyema will usually promptly remove the fever, as well as the night-sweats and the other symptoms of septic poisoning. The appetite and ability to digest food return with their disappearance. As the drain upon the system is great, the maintenance of strength is all-important. Food should be given as freely as the stomach's digestive power will permit. It must be easily digested and highly nutritious. In conditions of great weakness milk, eggs, custards, and beef-juice form the best diet. As strength returns it can be varied, and made more nearly that of a healthy person.

Often, before drainage is established, the system is so fully poisoned by absorbed septic matters that great enfeeblement of the whole body exists. The pulse may be very small, soft, and quick. Vomiting may be of frequent occurrence, and diarrhœa and night-sweating may be exhausting. If drainage and the consequent removal of the source of intoxication do not cause these symptoms to disappear, the case must be treated symptomatically. For instance, the cardiac weakness must be counteracted by digitalis or one of its congeners, and the vomiting and diarrhœa must be checked by the drugs usually employed to combat such symptoms.

Malformations of the chest which result from pleurisy can frequently be corrected. This is especially possible during the first third of life. They are corrected by dilating the lung and by bringing about a hypertrophy of its tissue. These objects can be accomplished by persistently exercising the crippled lung. Frequent enforced deep breathing is useful. Postural breathing is still more useful. For example, as a long and deep inspiration is being taken the patient may lean to the unaffected side, so that its lung cannot easily expand, while every opportunity is given to its crippled mate. A still better postural exercise consists in grasping, during complete expiration, the thigh on the unaffected side with the hand of the same side, and in the slow raising of the opposite arm from the side by swinging it outward and upward, as an inspiration is slowly and deeply made, until it is stretched as high as possible above the head. Other forms of postural respiration can be devised that are better suited to the location of certain deformities. The inhalation of compressed air is exceedingly useful. A residence in high altitudes will also help to expand the chest. But they should be combined with postural respiration. Running, mountain-climbing, and other exercises that especially lead to involuntary deep breathing and lung development are useful aids.

Prognosis.—The prognosis of pleurisy must depend upon its character and cause. For primary dry pleurisy the prognosis is favorable. For dry pleurisy at the apex, especially if it is subacute or chronic, it must be guarded, as tuberculosis is its usual cause. As a rule, perfect resolution will take place in serous pleurisy, especially if a large amount of fluid does not accumulate. By aspiration most serous pleurisies in which effusions have been extensive will undergo resolution. Purulent pleurisy very rarely recovers spontaneously. By surgical treatment most cases can be cured, and if it is applied at the beginning of the disease almost every case can be saved.

CHAPTER XVI.

PNEUMOTHORAX.

Causes.—Air rarely exists long in the thoracic cavities without exciting inflammation, which is accompanied by a fibrinous, or oftener serous or purulent, effusion.

Air may gain access to the pleural cavity from without through a wound, such as a stab or bullet wound, but oftenest it is admitted by perforation of a lung, which permits its air to escape. A broken rib may tear the lung and cause pneumothorax, though the chest-wall is not opened. More rarely, after adhesive peritonitis has glued the stomach or an intestinal loop to the diaphragm, they are perforated by ulceration, and permit air or gas to escape from them into the pleura. The lung is oftenest perforated by the rupture of a phthisical cavity. This rarely occurs, except in cases of rapidly developed phthisis, for, if the disease is chronic, either the pleura becomes greatly thickened over the vomica or the costal and visceral surfaces adhere and obliterate the thoracic cavity. Gangrene and abscess of the lung may lead to perforation of the pleura. Lung-tissue which is emphysematously distended rarely gives way, and produces a minute channel by which air can escape from the lung. Forced respiration, associated with violent physical exertion, may produce such a rupture, and, in extremely rare cases, seems to be the sole cause of pneumothorax. An empyema may ulcerate through the pleura, and the pus may escape by a bronchus. The hole in the pleura thus formed is rarely the cause of pneumothorax. It has been asserted that purulent and putrid fluids in the pleural sac may produce gas and an apparent pneumothorax. It is very doubtful if this ever occurs. A single opening may admit the air to the pleural sac, but often several do. Pneumothorax is very rare in both pleuræ.

Varieties.—If the channel by which air enters the pleural cavity remain constantly open, it is called "open pneumothorax"; if closed, "closed pneumothorax"; and if it is open during inspiration and closed during expiration, it is called "valvular pneumothorax." A permanent closure may be effected by a fibrinous exudate, which becomes, at least in part, organized; or it may result from compression of the lung. In closed and valvular pneumothorax the air within the pleura is under more than atmospheric pressure, and if an exit is afforded it will escape with a hiss. A valvular pneumothorax will be formed when the perforation through the pleura is an oblique one. The air of a closed and open pneumothorax varies in composition. Carbonic-acid gas is more abundant, and oxygen less, in the closed. Often air can gain access to only part of the pleural cavity, because of adhesions.

Symptoms.—The onset of pneumothorax is generally sudden. If it is due to rupture of the lung, as it commonly is, a pain is suddenly felt in one side. The feeling is described sometimes as like the breaking or tearing of something. It may be agonizing or moderately severe. It may be transitory or may persist for some hours. When persistent it is very like the pain of pleurisy, and may be due to inflammation. Rapidly after the pain begins dyspnœa of varying degrees of intensity arises. If the pleural sac quickly fill with air, the lung upon that side will partly or wholly collapse, and the heart may be displaced. The heart will beat rapidly, because of the interference with the pulmonary circulation which the collapse causes. Rarely the development of pneumothorax is accompanied by the symptoms of collapse. The temperature falls; the countenance is anxious and gray; the skin is cold, and often clammy; the lips are purplish; the pulse is small, soft, 120 or more per minute. Death may occur in a few minutes, or in a few hours, from the great disturbance suddenly caused to respiration and circulation. Usually death is not sudden. If it occur at all, it is after days or weeks. Recovery may take place, but does not do so commonly. The primary disease—which is usually phthisis, or abscess, or emphysema—causes death, or a complicating purulent pleurisy may. If recovery take place, obstruction to the ingress of the air occurs, a serous exudate replaces it as absorption progresses, and finally the hydrothorax may be relieved by treatment or spontaneous absorption. If the air in the pleural cavity is not large or fills only a section of it, absorption may take place without being followed by a serous exudate. Commonly pneumothorax provokes inflammation of the pleura and a complicating serous or purulent inflammation.

A diagnosis can only be made from the physical signs which pneumothorax causes. The affected side is distended. The intercostal spaces are wide or bulging. Respiratory movements are slight or wanting, while exaggerated on the other side. Vocal fremitus is usually absent. The resonance is great. Percussion sounds are loud and clear. They may be tympanitic, but usually are hyper-resonant only, because the distension of the thorax-wall prevents the production of a tympanitic note. The area of resonance will be much greater than usual if the whole of a pleural cavity is filled with air. The liver will be depressed if the right side is affected, and the heart may be crowded wholly into the right thorax while the left side is affected. If the heart is thus displaced, its pulsations will be seen and felt to the right of the sternum, and its sounds will be heard there and will be lacking on the left side. Auscultation may demonstrate an entire absence of respiratory sounds. Oftener they are audible, but have a characteristic metallic quality. There may be amphoric metallic respiration. This occurs when there is an open pneumothorax, or the sounds may be the usual bronchial or tracheal ones, which acquire a metallic quality when they are transmitted through the air in the pleura. Metallic râles or tinklings are often heard. If, while auscultation is practiced, a pleximeter is struck by the handle of a percussion hammer or other solid body, a metallic percussion sound will be heard. If, as is so often the case, an effusion as well as air is in the pleural sac, the usual signs of a pleural effusion will be found over the most dependent part of the thorax. The surface of the fluid will not be curved, as in ordinary pleural effusions, but will be horizontal. It will change with changes in the position of the body. Moreover, the height of the metallic sounds produced by the pneumothorax will vary with changes in the position of the body, because of the resulting change in the shape of the air-space. Sudden movements of the body may cause a metallic splashing.

Diagnosis.—A diagnosis is usually not difficult, if the physical signs are sought for. It may be difficult to differentiate between a circumscribed pneumothorax and a large, superficial pulmonary cavity. The former is oftenest in the lower part of the thorax, the latter in the upper. The intercostal spaces are usually wide or bulging in the former, and contracted or retracted in the latter. Vocal fremitus is slight or wanting in the former and may be strong in the latter. Cardiac displacement is indicative of pneumothorax.

Treatment.—The indications for treatment are: (1) to relieve pain while it exists; (2) to strengthen the heart, if collapse occurs; (3) to relieve dyspnœa; (4) to treat pleuritic inflammation that may complicate the pneumothorax.

The pain which the pulmonary rupture causes is usually transitory. If, as rarely happens, it is severe, one or two doses of morphine may be needed. Persistent pain is usually due to pleurisy. Morphine or codeia may be needed for its relief. Often counter-irritants and fomentations will answer as well.

If the patient is in a state of collapse, cardiac and diffusible stimulants are required. Ammonia by inhalation and by the stomach produces prompt but transitory effects. Camphor acts in the same way, by stimulating the heart, and can be given hypodermatically, in an oil solution. (See page 101.) Digitalis and strophanthus produce more lasting but less prompt effects.

The dyspnœa which is due to a sudden compression of the lungs and interference with the circulation may endanger life. Often those who have not suffered from dyspnœa before the pulmonary collapse occurs are at once overwhelmed and fatally suffocated, while those who are habituated to dyspnœa by other lung-lesions may not be endangered by a similar accident. Dyspnœa produced in this way cannot be easily relieved. Aspiration of the air within the pleural sac has been resorted to. It can do no good unless the pneumothorax is a closed one. During the first few hours, while the dyspnœa is most keenly felt, it is least likely to be closed. After three or four days a small perforation may close, and then the cavity may be aspirated of its air. Often, just as when liquid effusions are withdrawn, coughing is excited or thoracic distress produced by aspiration. A part only of the air can then be withdrawn. Indeed, it is undesirable to remove it all, for by so doing there is danger of stretching the weak lung, so that it will again rupture.

Empyema and serous effusions must be treated as they would be when there is no pneumothorax. The former should be drained as soon as possible. If the pneumothorax is a closed one, drainage may be followed with a partial expansion of the lung, and gradually a complete expansion. Under these circumstances, adhesive pleurisy may gradually obliterate the cavity, and the suppuration may cease. If the pneumothorax is open and the lung cannot re-expand, not only is drainage needed, but usually resection of the ribs, to produce contact of the pleural surfaces.

Rest should be enjoined from the first, that no bodily exertion may widen the rent or prevent its closing. As the only hope of recovery lies in the preservation of life until absorption of the air can be brought about or complicating pleurisy cured, foods must be judiciously administered.

Pneumothorax from penetrating wounds is often curable. The wound must be closed. The air may be partly aspirated, but will usually be absorbed. The danger is from pleurisy, but if the air that entered the pleura was clean, and not infectious, pleurisy will not supervene.

Prognosis.—If pneumothorax is due to ulceration through the diaphragm from the stomach or intestines, a fatal result must be expected, and treatment must be palliative. If it is due to rupture of an emphysematous lung, serous pleurisy often does not occur, for the air admitted to the pleura need not be infectious. In such cases recoveries have often been recorded. But rupture of a phthisical cavity will almost invariably admit to the pleura infectious matter and provoke empyema.

Pneumothorax is always a dangerous disease. Death occurs in one-fourth of all cases within a week; in about one-half within a month; and in the rest life may be prolonged for a year, and in a few recovery will take place.

CHAPTER XVII.

HYDROTHORAX.

Causes.—Hydrothorax is always a secondary lesion. It is a form of dropsy that is associated with general œdema. Commonly it is caused by Bright's disease, heart disease, or emphysema. It does not often develop until after dropsy of the subcutaneous tissue and ascites have appeared. It is usually bilateral, but the fluid commonly accumulates in one side to a greater extent than in the other.

Diagnosis.—By compressing the lungs it causes dyspnœa, and may produce fatal suffocation. The physical signs by which it must be distinguished are those of pleurisy with effusion. If the physical signs are carefully noted it cannot be confounded with any other disease. To differentiate it from pleurisy may be difficult. The latter is rarely bilateral, as the former usually is; the latter is not commonly associated with general œdema; there are no evidences of inflammatory fever in the former, such as are present in the latter. If the fluid is withdrawn from the pleural cavity it will be found to contain the largest percentage of albumen when it is of inflammatory origin. If its specific gravity is less than 1015, it is usually regarded as an effusion; if it is more than 1018, an inflammatory exudate.

The fluid of serous pleurisy, when examined microscopically, is found to contain blood-cells, especially white ones, and epithelial cells; the fluid of hydrothorax contains few or none of them. The latter often coagulates spontaneously after it is drawn from the chest, and the former rarely does.

Treatment.—Hydrothorax must be treated by removing its cause. If the kidneys, skin, and lungs can be made to eliminate water freely a pleural effusion may re-absorb, as other dropsies may. Often it threatens life, and immediate relief is demanded. It can be afforded by aspiration.

Prognosis.—Hydrothorax usually necessitates an unfavorable prognosis. Even if the fluid is temporarily removed from the thorax by aspiration, it may return, for the primary disease which causes it can seldom be removed.

SECTION II.

Diseases of the Heart.

DISEASES OF THE PERICARDIUM.

CHAPTER XVIII.

PERICARDITIS.

Anatomy.—An inflammation of the pericardium constitutes pericarditis. The anatomical changes wrought by it are precisely the same as those produced by inflammation of the pleura. Its varieties are also the same; there may be a dry pericarditis, a serous pericarditis, a purulent pericarditis, and chronic pericarditis. Its results are similar; it may undergo resolution; it may produce adhesions and even obliteration of the pericardial sack; it may, if purulent, ulcerate through the pericardium and thorax, or oftener into the pleural cavity or a bronchus, or through the diaphragm, or into an abdominal viscus. Pericardial inflammation may extend by continuity to neighboring structures and excite inflammation of the heart-muscle, or pleurisy, or peritonitis. Serous and purulent effusions may rapidly compress the heart and interfere with its work. Pericarditis in any of its forms, if not very extensive or very acute, may not greatly disturb the heart's action.

Causes.—Pericarditis is rarely a primary disease. The causes of primary or idiopathic cases are imperfectly understood. It is usually secondary to inflammation of neighboring tissues, as of the pleura; to acute and subacute rheumatism; to miliary tuberculosis; to eruptive fevers, and to pyæmia.

Symptoms and Diagnosis.—The symptoms of pericarditis are often obscured by those of the primary disease. In the mildest cases and in chronic ones there may be no subjective symptoms. In severe cases, and especially if a pericardial effusion is considerable, patients usually appear anxious, cyanotic, and dyspnœic; or the mind will wander, the delirium sometimes being active and other times passive. Stupor and even coma may exist. Pain is occasionally felt keenly in the cardiac region, but generally is not very noticeable. It may be sharp and lancinating, or oftener constant, and rather a feeling of fullnes, of tension, and oppression than pain. Tenderness is usually noticeable over the heart. Headache is often associated with pericardial inflammation. Occasionally a dry, hacking cough may be heard. The patients usually prefer to lie with the head and shoulders raised, or to sit upright. Dyspnœa varies in intensity. It is usually slight, except when a serous effusion is considerable. The dyspnœa is in part due to pressure upon the lungs by an enlarged pericardium, but is chiefly due to the change in the heart's action. The ventricles dilate and fill imperfectly. The pressure within the right ventricle and the pulmonary vessels is low, and the current through the latter is slow. This is provocative of dyspnœa. The same physical conditions exist in the left side of the heart and in the general circulation. There is, therefore, a small and soft pulse. The heart is rapid, and, if much weakened or irritated, may be irregular. The veins about the neck are distended. These circulatory derangements cause the mental symptoms that are sometimes observable. Fever is usually due to the primary disease. It may be increased, by pericarditis, or remain unchanged. If the fever is due wholly to pericardial inflammation, it is found to follow no regular type, and is not high. Slight pericarditis and chronic pericarditis may exist without causing fever. Vomiting and hiccough are sometimes symptoms. They may arise from unusual pressure on the diaphragm, or from an involvement of the vagus nerve in inflammation. The general circulation may be sufficiently interfered with to cause general œdema, and even ascites or hydrothorax. If death occur, it may be due to cerebral congestion, œdema of the lungs, or the primary affection. The symptoms of recovery are diminution of dyspnœa and disappearance of the characteristic physical signs. The heart is usually excitable long after the inflammation has subsided, or is quickened by mental and physical exertion. The duration of acute attacks is ten days or two weeks. Chronic ones may last many weeks or months.

A diagnosis is possible, but must be made from the physical signs. The præcordia usually appears prominent. The intercostal spaces may even protrude if the effusion is great. The apex beat is generally invisible if there is much effusion. Under the same circumstances it may not be felt, unless the patient leans forward or to the left, so that the heart can gravitate against the chest-wall. A shifting of the position of the apex beat is diagnostic. If with changes in the patient's position the beat moves from right to left or disappears and re-appears as the body moves backward and forward, it is evident that the heart is swinging loosely in a distended sack. In the beginning of pericarditis, before effusion has separated the pericardial surfaces, and in the fibrinous variety, a pericardial friction fremitus can sometimes be felt. Palpation demonstrates tenderness in the cardiac region. By percussion the area of dullness is found to be increased whenever there is an appreciable pericardial effusion. The latter first accumulates about the base of the heart. As it becomes greater it separates the heart from the thorax and pulmonary pericardium. The heart gravitates to the most dependent part of the area. The shape of the normal area of dullness is modified. It becomes more quadrilateral. It is broadened above, and, when the effusion is considerable, also below. When the effusion is great the dull area may reach the axilla to the left and the nipple to the right. Sometimes along the lateral borders of the area of dullness there is a semi-tympanitic resonance, which is due to the compression and consequently relaxed condition of the lung. If when the patient sits upright the apex beat is not in the left lower corner of the area of dullness, but nearer the median line, it is evident the pericardium must be distended with fluid. At the beginning of acute pericarditis friction-sounds can be heard in almost every case. They will disappear if effusion take place sufficient to separate the pericardial surfaces, but may re-appear as the fluid is re-absorbed. They may be absent in chronic pericarditis. The sounds are usually not loud, and can rarely be heard beyond the area of cardiac dullness. They are often loudest when the body is in certain positions, as when it leans forward, or when deep inhalations are drawn, or when the stethoscope is pressed against the chest. Sometimes they are heard only under these circumstances, and are not constant. Usually they are hitching in character. One sound is heard with the contraction of the auricles, and is presystolic; another with the systole, and a third with the ventricular expansion, or diastole. The character of the sounds varies. They may resemble a soft rub, or be creaking. They may be so loud as to obscure the cardiac sounds. Usually the latter are plainly audible, unless an effusion makes them low and distant. Endocardial murmurs may be heard simultaneously with the pericardial sounds, and may be due to blood-states or to endocarditis.

A diagnosis of fibrinous pericarditis can only be made when pericardial friction-sounds are heard. They are pathognomonic. Endocardial murmurs need not be mistaken for pericardial, for they are blowing, not rubbing, are not increased when the stethoscope is pressed against the chest, or during deep inspiration, and are always synchronous with a given part of the heart's cycle. It is never difficult to distinguish extra-pericardial or pleuro-pericardial friction-sounds from them. Pleuritic and pericardial friction-sounds may be heard together, both being produced in the pleural cavity, and they will cease when breathing is stopped. The absence of subjective symptoms, which may accompany pericarditis, will help to confirm a decision.

Permanent pericardial adhesions, or a partial or general obliteration of the pericardial cavity, may exist for years. They may result from acute or chronic inflammation. If the adhesions are small, they are oftenest near the large cardiac vessels. The heart-muscle underlying the adhesions is usually involved in connective-tissue infiltration and degeneration and atrophy of the superficial muscle-fibres. A diagnosis of adhesive pericarditis, even when it is extensive, may be impossible. In some cases the adhesions constrict the coronary arteries and interfere with the heart's nutrition. The symptoms then are of heart-fatigue, or exhaustion. The concurrence of two symptoms may be considered positive evidence. These are a systolic retraction of the apex, intercostal spaces about it and the lower end of the sternum, and diastolic collapse of the cervical veins. A systolic retraction of the apex alone may be produced whenever the heart cannot be pushed downward during systole. Rigidity of the arch of the aorta, as in atheroma, may cause it; aortic-valve stenosis may; and even slight pericardial adhesions at the base of the heart may. If the apex cannot descend during systole, it cannot produce the normal beat, for the ventricles, when they contract and lengthen, push the apex backward, instead of forward, and thus cause retraction. But if the retraction is diffuse and involves several intercostal spaces and the lower part of the sternum, it is quite surely due to pericardial adhesions. When systolic retraction occurs, a diastolic apex-beat may be produced. The diastolic projection of the chest-wall which is thus produced probably favors the indrawing of the venous blood by the heart, and therefore a diastolic collapse of the cervical veins. The diastolic projection may also cause a dull sound to follow closely, and seemingly duplicate the diastolic sound of the heart.

The existence of pericardial effusion can usually be diagnosed directly from the physical signs that have already been described. The increased area of dullness which it causes may have to be differentiated from (1) enlargement of the heart, which is accompanied by an apex-beat, in the lower left corner of the dull area, and by greater distinctness of the heart's sounds; from (2) mediastinal tumors, aneurisms, encysted pleuritic effusion, and consolidation of the borders of the lungs adjacent to the heart, which produce a greater irregularity of outline of the dull district and characteristic general and local symptoms. The apex-beat is lacking in pericardial effusion; at least, it is lost when the patient lies upon his back. It is also often absent when the heart is weak, and not hypertrophied. In the latter case the cardiac sounds are not distant, and the outline of the dull area does not change with the position of the body. It may be absent when the borders of the lungs are emphysematous, but the area of cardiac dullness is then small.

The character of a pericardial exudate can only be told by aspirating a part of it. If pyæmia is the cause of pericarditis, it may be purulent. If rheumatism is, it probably is serous. Tubercular pericarditis is rarely primary. It may be acute or chronic. The symptoms in the following case will illustrate those of many: A young man, aged 24, entered Mercy Hospital, after an obscure illness of two weeks. He was slightly dyspnœic. His pulse was feeble and quick. He often felt apprehension. At times he was nauseated. There was a trifling œdema about his ankles. His temperature was very irregular, but never high (102° F. and less). He had colliquative sweats. The physical signs of pericardial adhesions were plainly present. At first no tubercular lesion could be found in the lungs, but later they were involved. Some months after the beginning of this illness he died from pulmonary phthisis and cardiac weakness. Several of his brothers and sisters are tuberculous or have died of phthisis. In more acute cases a suddenly developed cardiac fatigue produces the existing symptoms.

Slight pericarditis often undergoes resolution. It is, however, always a grave disease. Purulent pericarditis is almost certainly fatal.

Treatment.—The indications for treatment are to remove the primary affection as promptly as possible, and in its early stages to combat the inflammation by rest, by derivatives, by depletors, and by opiates. Rest should be enjoined, at least so long as acute pericarditis or pericardial effusions exist. Blisters, cups, and leeches applied over the heart often lessen the severity of the general symptoms which may arise. A promptly acting cathartic is often prescribed as a depletor. By these agents inflammatory congestion may be lessened and the extension of inflammation prevented. Opiates are useful when pain and tenderness are great. Calomel, in doses of from 0.015 to 0.06 gramme (¼ to 1 grain), is often prescribed to hasten resolution, and especially the prompt absorption of serous and fibrinous exudates. If heart-fatigue or exhaustion threaten, cardiac tonics and stimulants must be relied upon to maintain the heart's vigor until rest, nourishment, and the removal of the cause of weakness will effect a permanent restoration of it. Digitalis, strophanthus, convallaria, caffeine, ammonia, and camphor may be used. (See page 222.) To remove the effusion, blistering plasters may be applied over the heart. Often diuretics and diaphoretics are relied upon. Of the former, digitalis, strophanthus, potassium, and ammonium acetate are commonly used. Dry heat is chiefly employed to provoke sweating, for preparations from jaborandi weaken the heart's action. These same drugs must be used to remove dropsies which pericarditis may cause.

If cardiac exhaustion is threatened by an effusion, it may be averted by aspirating the fluid. Aspiration gives prompt relief, but it is often only temporary, as the effusion may re-form. The needle should be inserted in the fourth or fifth intercostal space, close to the sternum. If the liquid is pus, permanent drainage of the pericardial cavity should be established. Recoveries have occasionally been obtained by this treatment. After pericardial adhesions have formed they cannot be influenced by drugs.

CHAPTER XIX.

HYDROPS PERICARDII.

HYDROPS PERICARDII may be produced under the same circumstances as hydrothorax. (See page 194.) Compression of the coronary veins, for instance, by pericardial tubercles or cancerous nodules, may cause it. It will cause the same symptoms as pericarditis with liquid effusions, except that friction-sounds will not at first be present. It can be distinguished from the latter by its causation and by the low specific gravity of the fluid (below 1015), by its usual association with other dropsies, and absence of pericardial tenderness. Aspiration may be practiced, and even often repeated, when compression of the heart threatens its exhaustion. Diuretics and diaphoretics are used, as in other dropsies, to promote absorption of the fluid exudate. If cardiac exhaustion is imminent heart-tonics must be used, as in pericarditis. Complete absorption of the fluid and recovery are possible; but pericardial dropsy may re-form after such absorption if its cause is not removable.

CHAPTER XX.

PNEUMOPERICARDIUM.

Causes.—Pneumopericardium may be caused by (1) external, penetrating wounds; by (2) contusions of the thorax, which cause the fracture of a rib, that in turn lacerates both the lung and pericardium; by (3) ulceration of tubercular or gangrenous pulmonary cavities into the pericardium; by (4) ulceration of the œsophagus or stomach, either simple or cancerous, into the pericardial sack, and by (5) ulceration of a purulent pericarditis into the œsophagus, a bronchus, or the stomach. By most of these processes pyogenic matter will be admitted to the pericardial cavity, and purulent inflammation, as well as pneumopericardium, will be the result.

Symptoms.—Subjective symptoms which accompany the lesion are usually as obscure and as little characteristic as in pericarditis. If much air fill the pericardial sack, heart-exhaustion will rapidly be produced.

The physical signs are characteristic. On inspection the præcordia usually seems prominent and the intercostal spaces protrude. The apex-beat is invisible. Palpation will not reveal it, unless when the patient leans forward the heart gravitates against the chest. Sometimes a friction-fremitus can be felt, or a peculiar splashing. On percussion the area of cardiac dullness is replaced by unusual resonance. A metallic quality is imparted to the percussion sounds if a pleximeter is struck with a rod or metallic body. Auscultation reveals, also, a metallic quality of the cardiac sounds which they do not normally possess. If fluid is also in the pericardium, they will only be heard when the patient is recumbent. If friction-sounds are audible, they, too, will be metallic in character. Fluid and air both within the pericardium usually produce, when stirred by the heart's movements, splashing or gurgling sounds.

These various metallic sounds, as well as areas of resonance that in part displace the heart's dull area, may be produced by large, superficial cavities in the lungs beside the heart, through which its sounds are transmitted, or they may be simulated by the stomach when it is greatly distended upward.

Treatment.—The treatment must be symptomatic. Cardiac tonics are indicated if heart-exhaustion threaten. Pain must be allayed by opiates. If the gas within the pericardium is dangerously compressing the heart, it may be aspirated out. If purulent inflammation occur, drainage should be established. We may attempt to limit the inflammation by blisters, cups, and leeches, or by the ice-bag, as in simple pericarditis.

Prognosis.—The prognosis must be unfavorable unless the pneumopericardium is caused by a penetrating wound of the thorax which has not admitted pyogenic matter.

DISEASES OF THE HEART-MUSCLE.

CHAPTER XXI.

DILATATION OF THE HEART.

Causes.—Dilatation of the heart may be secondary to other cardiac lesions, as, for instance, valvular ones, or degeneration of the muscles; or it may be secondary to arterial lesions and to diseases in distant organs. The latter are often called idiopathic cases. The causes of dilatation of the heart are mechanical and nutritive. Frequently both act together in a given case. Of the mechanical causes those oftenest acting are valvular lesions of the heart; obstructions to the aorta by compression or by contraction; aneurisms of it; arterial sclerosis, not alone of the aorta, but of the smaller arteries as well. Obstruction to the pulmonary circulation will provoke dilatation of the right side of the heart. This may be caused by chronic disease of the lungs, which destroys many of the capillaries and, it may be, some of the larger vessels. Pleuritic effusions which will compress a lung will also interfere with a perfect pulmonary circulation. Sudden, severe physical exertion will occasionally cause cardiac dilatation; but this will rarely happen unless in some way the heart's muscle has been previously weakened. Most of these mechanical causes produce dilatation of only one of the cavities of the heart. A few affect it more extensively.

If nutrition has been imperfectly maintained in the muscle of the heart, it may be dilated by an exertion which is not abnormally great. Malnutrition oftenest results from fevers,—especially protracted ones,—from anæmias, from indigestions which cause a general malnutrition, from fatty degeneration and infiltration, and from obstructions to the coronary circulation.

Anatomy.—Dilatation of one ventricle only may occur, as when there is aortic-valvular insufficiency; or both sides may be distended, as in mitral insufficiency. Usually, dilatation and hypertrophy co-exist, but the ventricular walls may be thin. Often the heart is pale from degeneration of its muscle. Venous hyperæmia may exist in many organs if the dilatation has been chronic or great.

Symptoms.—A diagnosis can only be made from the physical signs which dilatation of the heart produces. In many cases there are no subjective symptoms that can be ascribed to the heart-lesion, although there may be others that are due to a primary affection.

If there is no hypertrophy of the heart accompanying the dilatation, the apex-beat will be invisible, or it will be feeble and diffuse. It is displaced more or less to the left. On palpation the heart's beat can usually be felt, even when it is invisible. If much hypertrophy accompany the dilatation, the apex-beat may appear strong and feel lifting and energetic; but the pulse will be small, soft, and quick. Irregularity of the heart is often due to dilatation.

Percussion will demonstrate an increased area of cardiac dullness. The directions in which this increase is greatest will depend upon whether a single cavity of the heart or several are involved. Auscultation will reveal feeble cardiac sounds. The first sound will lose much of its booming character and resemble closely the second. Even when valvular lesions do not exist systolic murmurs may be heard. This will rarely happen except when dilatation is associated with anæmia. If hypertrophy is co-existing the sounds may be booming, and even stronger than natural. We can, then, conclude that dilatation exists only because there are evidences of heart-exhaustion, and we know that where they exist with hypertrophy dilatation does also. With valvular lesions we know that dilatation and hypertrophy are always associated.

Subjective symptoms may exist, although commonly they are wanting. If the dilatation is great the usual symptoms of cardiac exhaustion will be present. They result chiefly from an imperfect balance of the venous and arterial blood. The arteries grow small and the veins dilate; passive engorgement, therefore, develops. Dyspnœa, enlargement of the liver and spleen, the symptoms of renal engorgement, or general dropsy may exist alone or in combination.

In mild cases, and especially if the dilatation accompany an excitable nervous system, the quick beating of the heart may be felt, as palpitation. A beat may drop or be delayed, and may then cause anxiety or fright. Frequent and uncontrollable sighing so often accompanies dilatation of the heart that I always search for the latter when I observe the former. The sighing is undoubtedly due to an unsteady enervation of the respiratory muscles; it may also be caused by mental depression or flatulent indigestion. Not unfrequently one of these latter conditions accompanies cardiac dilatation. A lack of energy and endurance characterizes all cases of cardiac dilatation, unless compensating hypertrophy co-exists.

Treatment.—The object of treatment is to cause contraction of the heart or compensating hypertropy of its muscle. Strength may be temporarily given by the drugs that increase the force of the systole, such as digitalis, strophanthus, and convallaria. It can be permanently maintained only when any degeneration of muscle-fibres that may exist undergoes resolution, and when with suitable tonics, foods, and exercise they are invigorated. The tonics which will stimulate the cardiac tissues to a better degree of nutrition are strychnia, caffeine, quinia, and iron. They are indicated not only when anæmia exists, but also when muscular degeneration does. They may be best given combined with digitalis or its congeners.

If it is a moderate dilatation arising from a protracted fever that must be dealt with, sponging the skin and baths with tepid or cold water stimulate the circulation and aid in maintaining a vigorous action of the heart which will prevent dilatation.

If the heart is dilating, liquids must not be drunk in quantities sufficient to augment the blood. This is especially true if there is dropsy or anæmia. Then a reduction of the bulk of the blood will prove advantageous. (See page 224.) Food must be nutritious and not difficult to digest. Martin has shown that alcohol will cause dilatation of the heart. Such beverages certainly predispose to it by leading to tissue degeneration and to loss of vascular and cardiac tone. They should be excluded from the dietary of those suffering from this disease.

If moderate exercise is steadily persevered in it will lead to a better nutrition, and, it may be, to hypertrophy of the heart's muscle. If it is violent, it may strain the muscles by unduly increasing the blood-pressure, and thus augment the dilatation.

Antipyretics diminish tissue-change and interfere with nutrition. Their frequent or prolonged use is contra-indicated whenever degeneration exists and we wish to prevent its spread. Aconite, veratrum, and other drugs that cause vascular and cardiac relaxation are contra-indicated.

Prognosis.—The prognosis of cardiac dilatation will depend entirely upon the possibility of removing its cause. Mechanical causes usually cannot be removed. If the obstruction which they cause to the circulation can be overcome by hypertrophy, they will not endanger life. Dilatation from a weakness of the muscular fibres of the heart, because of their malnutrition, can generally be perfectly overcome. If degeneration is extensive or its cause cannot be removed, a favorable result is impossible. Weak muscles, because of hæmorrhage, acute or subacute anæmia, indigestion, or fever, is curable; but if it is due to chronic Bright's disease or to obstruction to the coronary arteries, it is incurable.

CHAPTER XXII.

CARDIAC HYPERTROPHY.

Anatomy.—When the heart's muscle hypertrophies, the number of fibres may increase or they may enlarge. Usually, both these changes occur simultaneously. Hypertrophy may enlarge the entire heart, or a part of it. A few of the muscular papillæ only may enlarge, or the right or left ventricle, or both. The auricles are capable of very limited hypertrophy. The cavities of the heart are usually dilated when the walls hypertrophy, but may remain normal, or, in extremely rare cases, be contracted. The interstitial tissues sometimes increase. If, in spite of the hypertrophy, the heart is continuously fatigued, it may undergo fatty degeneration. Hypertrophy causes the heart's wall to appear thick, and to be unusually firm and hard. It is usually normal in color, but may be a brownish red, from an excess of pigment in it.

Causes.—Hypertrophy is the result of prolonged overexertion. This may be due to obstruction to the circulation, which must be overcome, or to unusual and prolonged muscular work, and rarely to nervous excitement and strain. Obstructions may exist within the heart at its valvular orifices,—for example, in chronic valvular disease,—or outside the heart, as in stenosis or compression of the aorta or small arterioles. Endarteritis obliterans often obstructs the arterioles extensively, as in the kidneys in chronic interstitial nephritis. Aneurisms of the aorta or its main branches cause additional work for the left ventricle, and provoke it to hypertrophy. Permanent pericardial adhesions frequently lead to hypertrophy. Acute and chronic parenchymatous nephritis are sometimes accompanied by cardiac hypertrophy. It cannot always be due to vascular obstruction in these cases, for the latter does not always exist. It is probably sometimes due to a chemical cardiac irritant which is in the blood.

Symptoms.—Hypertrophy of the heart is usually a secondary lesion. Subjective symptoms that accompany it are due to the primary affections. Occasionally persistent hard beating of the heart is felt, and especially during left decubitus. But one must depend upon physical signs in order to make a diagnosis. The præcordia is unusually prominent. Particularly is this true in the earlier years of life. The apex-beat will be seen to the left of the nipple, and lower than is normal. The beat moves a larger area of the chest-wall than is usual, and it is more powerful and lifting. Palpation confirms its location, its diffusion, and its strength. If the left ventricle only is hypertrophied, its anterior surface will press against the thoracic wall, and the right ventricle will be rolled backward. The apex-beat will then be produced by the left ventricle. If the right ventricle is chiefly or exclusively hypertrophied, pulsation may be seen and felt to the right of the sternum, and will, in almost every case, be easily demonstrable just beneath it. This is due to the contact of the right ventricle with the thoracic wall, for when it hypertrophies the left is displaced backward, and the right ventricle chiefly forms the anterior surface of the heart. Percussion demonstrates an enlargement of the area of cardiac dullness. If the left side of the heart is exclusively involved, it will be increased to the left; if the right side, to the right; and if both sides, in each direction. It may extend an inch or more to the left of the nipple, and the same to the right of the sternum. The cardiac sounds are usually normal. The first sound at the apex may be louder and more booming. If the left ventricle is hypertrophied, the second sound over the aorta is accentuated. If the right ventricle is hypertrophied, it is the second sound over the pulmonary artery that is accentuated. These accentuations are due to increased tension of the respective semilunar valves. If both sides of the heart are thickened, we find a combination of the signs just described, and especially evidence of cardiac enlargement, both to the right and left. The cardiac sounds can often be plainly heard at a considerable distance from the heart. The carotids often pulsate visibly, and sometimes a systolic murmur can be heard in them which is due to the unusual tension of the vessel's wall and its consequent irregular vibrations. The pulse will be large and firm.

If the cause of the heart's increased work cannot be removed, or at least compensated for, the heart will become fatigued, in spite of its hypertrophy. It will grow rapid in action, or irregular. The lungs will become congested, and dyspnœa will develop. The liver will enlarge. The legs may become œdematous. The urine will diminish in quantity and may grow cloudy and contain albumen. Dropsy of the abdominal cavity or other serous sacs may be produced. The pulse will be soft, of medium or small size, in spite of the strong throb of the heart against the chest. Death may be caused by œdema of the lungs or heart-exhaustion.

Moderate hypertrophies which are produced by causes that are removable may disappear when their cause is gone.

Hypertrophy is a conservative process. It is favorable to life. It may enable the heart to overcome, perfectly and with ease, a permanent obstruction to the circulation. Unfortunately, often the obstruction gradually increases, and finally cannot be compensated for by hypertrophy. Then the symptoms of cardiac exhaustion develop.

Treatment.—No treatment is indicated if hypertrophy compensates for an existing obstruction to the blood's flow; but after compensation has been obtained the heart must not be wearied by additional and unnecessary labor. Therefore, fatiguing exercise must be avoided. Constipation and slow or labored digestion will impede the circulation and involve the heart in extra labor. They must be prevented or promptly cured. The diet should be simple and nutritious. Stimulants and tea, coffee, and tobacco must be avoided. Milk, eggs, lean meat, simple vegetables, and wholesome fruit may be used. Greasy, fat, and very farinaceous foods must be used sparingly, or not at all. Sufficient exercise in the fresh air should be taken to maintain a good oxygenation of the blood and a fair degree of muscular vigor, but it should never be exhausting or violent.

The nitrites are sometimes used (see page 228.) when high arterial tension is the cause of hypertrophy. They will lessen it, and thereby reduce the heart's labor. Digitalis is contra-indicated so long as the heart beats slowly and steadily. If it is often or persistently quick or irregular, it may be used. Strophanthus is to be preferred because, in usual doses, it does not cause so much arterial contraction, and, therefore, as high arterial tension. The former gives the heart more work to do. After the heart has hypertrophied, and then grows rapid or irregular in action, we may feel sure that dilatation is forming or increasing, and, usually, that fatty degeneration is established. The treatment must then be adapted to combat these lesions. Cardiac exhaustion is the cause of death when the heart is hypertrophied. Its prevention is, therefore, *the* indication for treatment.

CHAPTER XXIII.

FATTY HEART.

Anatomy.—Two lesions are named fatty heart. They are technically named fatty infiltration and fatty degeneration. In *fatty infiltration* the connective tissue beneath the pericardium, and especially about the coronary vessels, is filled with fat. The entire heart may be thickly enveloped in it. Fat may also accumulate between the muscle-fibres in the fibrous frame-work of the organ. The muscular fibres may atrophy and become very small. Such an accumulation of fat and concomitant muscular atrophy interfere with a vigorous action of the heart. As the lesion is almost limited to obese people, the cardiac fatigue which often co-exists with it is partly due to the larger volume of fluid that must be moved through their more numerous capillary vessels.

Fatty degeneration is due to a malnutrition of the cardiac muscle which may lead to its disorganization. In the muscle-fibres appear minute granules, that often obscure their striated structure. If the malnutrition is sufficiently great, the outline of the fibre is lost. A crowd of granules represent it for a time, but soon they are absorbed. Thus, disintegration and disappearance of tissue may grow out of the degeneration. The muscles that are affected are always weakened. They show this by a lack of endurance, as well as by a feebleness of contraction. The entire heart and many other organs and tissues may be simultaneously affected in this way; but usually only a patch of muscle-fibres here and there is involved. The muscular papillæ and the fibres beneath the endocardium are especially apt to be. The inside of the ventricle often appears mottled with yellowish spots. When the entire heart is degenerated it will all look yellowish-red and greasy. It will be soft. If a knife is drawn across the cut surface of degenerated muscle-fibres, droplets of oil can be seen in the fluid that gathers on it. Cloudy swelling may precede fatty degeneration, or be associated with it.

Symptoms and Causes.—A positive diagnosis of fatty heart is difficult to make, and is often impossible. Fatty infiltration may be suspected in persons who are obese and whose heart's action is feeble. The thick chest-wall may make it impossible to say whether the heart is enlarged or not, or to judge of its strength by the apex-beat, for the latter often cannot be felt. But if the heart is weak it will beat fast, even from moderate physical exertion. Its first sound will be short, and valvular in character. The pulse will be small and soft. If the heart is much enfeebled, the cervical veins may be distended. Shortness of breath is partly due to the cardiac weakness, and largely to the obesity of the chest. A feeling of oppression is often experienced, and sighs for breath, which are unsatisfying, are involuntarily drawn. Little endurance is possessed. A disinclination for physical exertion is usual.

Much fatty infiltration or fatty degeneration may exist and be unaccompanied by symptoms. Spontaneous rupture of the heart has been known to occur suddenly, because of the weakness of its walls. In other cases the symptoms of heart-exhaustion may develop, such as chronic venous hyperæmia and œdema of various organs; or angina pectoris may develop. A slow pulse, pseudo-apoplectic attacks, and Cheyne-Stokes respiration, if they occur together in the same person, are quite characteristic of fatty heart. Unfortunately, they rarely occur together, and either symptom alone is not characteristic. The pulse may be very slow,—even less than twenty beats to the minute. This is not, however, usual, for oftener it is quickened, at least, by bodily exertion. The arcus senilis frequently develops in those in whom fatty degeneration exists extensively, or results from local anæmias that, in turn, are caused by arterial stenosis. The presence of some of the causes of fatty degeneration aid one to make a diagnosis. Prolonged anæmias are especially apt to produce such degeneration. Chlorosis, leukæmia, and pernicious anæmia are examples of the forms oftenest leading to it. Chronic or protracted fevers will also cause it. Valvular lesions or arterial obstructions which cannot be compensated for by hypertrophy are common causes. Coronary sclerosis, by producing local cardiac anæmias, may lead to it. Phosphorus poisoning will cause intense general fatty degeneration. Chronic tobacco and alcoholic poisoning may lead to similar results. Obesity and fatty infiltration of the heart-muscle may be an inheritance; oftenest it is due to a lack of vigorous exercise and an excess of fat-producing foods.

Treatment.—If the heart beats feebly and is excitable, because of fatty degeneration, cardiac and general tonics are indicated. Anæmia requires iron. This drug also seems to prevent degeneration. Such general tonics as strychnia and quinia will invigorate nutrition. Such cardiac tonics as digitalis, strophanthus, and caffeine are required to temporarily strengthen its beats and restore the equilibrium of the venous and arterial currents. Strophanthus is preferable to digitalis in these cases, for it contracts the peripheral vessels less and increases the arterial tension less. Caffeine seems not only to stimulate the heart, but to increase its ability to appropriate nutriment. A combination like the following will often prove promptly efficacious:—

℞ Ferri citratis, . .	grms.	12.0 (gr. ij).
Caffein. citratis, . .	grms.	15.0 (gr. iiss).
Pulveris strophanthi, .	grm.	0.015 or 0.02 (gr. ¼ or ⅓).

Sig.: To be given in a capsule every four hours.

It is especially adapted to the stage in which palpitation is easily provoked. As the heart grows stronger, strophanthus or digitalis may gradually be omitted and strychnia or quinia substituted. It must be remembered of powdered strophanthus that it is laxative. Such treatment must be persisted in for weeks, and often for months. Inhalations of oxygen have been recommended, as a lack of it is supposed to cause degeneration. But we have no evidence that the blood will take up more oxygen if it is breathed pure than when diluted, as in common air. Respiratory gymnastics (see page 151), which will insure frequent emptyings of the lungs and their complete expansion with fresh air, will accomplish quite as much as oxygen inhalations. To maintain good nutrition of the heart good nutrition must be maintained everywhere. A perfect lymph-circulation is essential for this, and can only be assured by general exercise. Exercise should be gentle, but should be as long continued as is possible without producing a feeling of excessive fatigue or exhaustion. It should not be violent.

To diminish the amount of fat which may infiltrate the heart's muscle in obese persons, Oertel's treatment is the best. It aims to lessen the bulk of fluid in the vessels and the addition of fat, and to strengthen the heart by exercise. If the quantity of blood and fatty tissue is lessened, the amount of work which the heart has to do, in moving the fluid through the adipose tissue, is diminished. Copious sweatings will relieve the system of its excess of liquid, but to prevent its prompt restoration its ingestion must be limited and carefully prescribed. Sweating may be produced effectively by Turkish baths, or by vigorous and prolonged exercise. To prevent the continued accumulation of fat, carbohydrates must be eaten very sparingly. If an albuminous diet is adhered to, the fat already stored in the body will be utilized as carbohydrate food. This is especially true if exercise is taken freely, so that tissue-changes are kept vigorous. Exercise will provoke sweating, will maintain a vigorous circulation, necessitate deep and frequent breathing, thorough blood oxygenation, and active tissue-change; it will strengthen voluntary muscles and cause them, and with them the heart, to hypertrophy. Exercise should, if possible, be continued for several hours daily, by those who need this treatment. It should gradually be increased. Oertel recommends mountain-climbing more than any other form. In level countries, rapid walking, and, later, as strength and endurance increase, running may be substituted for climbing. It should be sufficiently active to provoke some shortness of breath and increased rapidity of the heart's action. If these symptoms begin to cause distress, a few moments' rest should be enjoined. The amount of exercise that should be taken by an individual will depend upon its effects. Therefore, each patient must be closely watched and guided. Too violent exertion might permanently and dangerously injure the heart, which is already injured and fatigued.

When the quantity of fluid that is to be ingested must be prescribed, it is best to ascertain about how often and how much the patient habitually takes, and at first lessen the amount rather than the frequency of its use. So it is best to learn what his usual diet is, and then eliminate from it the greater part of the carbohydrates.

Alcoholics should be excluded from the diet of those who have fatty heart. They make tissue-change slow by lessening the oxygen-carrying power of the blood. They lessen the vigor of nutritive changes partly in the same way, and by expanding the peripheral vessels and slowing the peripheral blood-current. The weaker preparations, such as beer, are taken in such amounts as also to greatly augment the fluid within the body.

If the heart is exhausted and can be spurred only temporarily, the symptoms of passive engorgement and œdema of various tissues will be little affected, and, sooner or later, in spite of these drugs, the pulse will grow smaller and quicker. As the symptoms of exhaustion intensify, diffusible stimulants like ammonia and camphor are employed advantageously. (See page 101.)

To relieve the œdema which may accompany fatty heart, such as anasarca, ascites, pleural and pericardial dropsy, the alkaline diuretics and more or less drastic cathartics (see page 292) can be used in addition to cardiac tonics. The serous cavities may have to be aspirated or punctured to effect immediate relief.

CHAPTER XXIV.

INDURATIVE DEGENERATION.

Anatomy.—By indurative degeneration I mean a lesion that is primarily a degeneration, and secondarily an hyperplasia, of connective tissue which causes induration. It results from a gradually produced and persistent local anæmia within the heart. Sclerosis, thrombosis, or embolism of some branches of the coronary arteries are its usual causes. In the anæmic area there is first degeneration and atrophy of the muscle-fibres, because of diminished nutriment and a lessened vitality. Under conditions of nutrition, in which highly specialized structures, such as muscle-fibres, cannot live, the connective tissues grow. This is possibly nature's method of attempting to repair the weakened fabric. If a heart in which induration and necrosis has developed is examined, there will be found imbedded in the muscle, and usually near the apex, a patch of gray, fibrous tissue. The heart's wall may or may not be very thin at this point. The patch may be minute, or the size of a half-dollar. While oftenest in the ventricular wall, and near the apex, it may be anywhere in the heart's substance. It may be deeply imbedded in the muscle or near its surface. It may involve only a small part of the heart's wall or its entire thickness.

Under the microscope the indurated tissue shows its connective-tissue character. If the patch is still growing, about its margins the cells composing it will be embryonic in type, and will infiltrate the neighboring muscle-fibres, which will be granular and atrophied. The result of these anatomical changes is always the production of a hard, cicatrix-like structure. But the loss of muscle-fibres makes the wall of the heart weak and the connective tissue cannot compensate for the loss. Rupture of the heart through the indurated tissue may occur, or, if the latter is just beneath the endocardium, and roughens it, a cardiac thrombus may form, which, in turn, may produce emboli. Cardiac aneurism is a third occasional result of this lesion. At the point of weakness the heart's wall will bulge and form a thin-walled sac containing blood. Such aneurisms may be the causes of thrombosis, because of the slow blood-stream within them, or they may rupture.

Symptoms.—Indurative degeneration may exist and produce no symptoms. In some cases the only symptoms are those of cardiac fatigue or exhaustion. The group of symptoms named *angina pectoris* are common accompaniers of this lesion. Unfortunately, they may also occur with fatty degeneration of the heart, with coronary sclerosis without indurative degeneration, with aortic valvular lesions, and aortic aneurism. But though this is true, angina pectoris is always suggestive of coronary sclerosis, and the latter of indurative degeneration. If with angina pectoris we find evidence of sclerosis of radial or temporal or other arteries, we may feel quite sure of coronary sclerosis.

Angina pectoris implies, when it is severe, an agonizing pain of oppression in the chest, about the sternum, accompanied by radiating pains to the left breast and shoulder, and into the left arm. The sufferer's face expresses pain, and even fear. The skin is pale, cold, and often clammy. The pulse is small, hard, and quick. The heart-beats are rapid, feeble, and often irregular. More rarely they are diffuse and vigorous, though the pulse is weak or irregular. Respiration is oppressed, irregular, sighing and unsatisfying, but not dyspnœic. Death from heart-failure may occur in the midst of such an attack. Usually the onset is not sudden, but, from a feeling of discomfort during two or three hours, an agonizing pain develops. It may last a few moments or for hours. When it subsides, vomiting not unfrequently occurs. When the pain is gone, as a rule, great prostration remains. The pulse gradually grows full and slow, and often is irregular and excited by slight physical or mental exertion. The attacks may recur frequently,—at least, every day or two,—or at long intervals of months or years, or not at all. They may be of all grades of severity, from slight, almost momentary, attacks of heart anguish to those of intense severity.

It is evident, however, that during life a positive diagnosis of indurative degeneration is impossible, for neither the symptoms of cardiac fatigue nor angina pectoris are pathognomonic of it. If angina pectoris and arterial sclerosis co-exist, we may conclude that the former is due to coronary sclerosis, and we know that this may lead to indurative degeneration. In rare cases angina pectoris occurs when no lesion of the circulatory apparatus is demonstrable.

Treatment.—Treatment must be symptomatic. If there is cardiac fatigue or exhaustion, it must be treated as in other diseases. (See page 222.) Angina pectoris may be relieved by morphine, or chloroform, or ether; but these drugs must be used with great caution, as they are liable to produce functional derangements of the nervous system. The nitrites are equally efficacious in certain cases, especially in those in which there is an arterial spasm. Amyl nitrite may be given by inhalation with wonderfully prompt effects. The cold, gray skin of the face soon grows flushed and warm, the head feels full, and the agonizing breast-pain lessens. Nitroglycerin is equally beneficial in the same cases, but little less promptly so. It may be given in doses of 1 or 2 drops of a 1-per-cent. solution, and may be repeated every three or four hours. The nitrite of soda may also be used, in doses of 0.06 to 0.12 gramme (1 to 2 grains) of the pure drug, or 0.3 to 0.45 gramme (5 to 8 grains) of the preparation usually dispensed.

While the patient is cold he should be made warm with hot flasks and his skin chafed. To avoid returns of the attacks, the patient should eschew excitement or undue bodily exertion,—should in no way produce high arterial tension by arterial contraction, as may be done by constipation or indigestion.

Such hygiene as will promote good nutrition, active tissue-oxidation, and healthful muscular vigor will help to prevent these attacks when a degenerating or enfeebled heart causes them.

CHAPTER XXV.

MYOCARDITIS.

Anatomy.—Indurative degeneration has been by many regarded as of inflammatory origin. But inflammation in the heart-muscle is due to extension from neighboring tissue or to septic infection. It commonly results from a peri- or endo- carditis which deeply involves the heart-wall. Inflammation may produce thickening of these thin tissues and destruction of the superficial muscular fibres by causing their atrophy or degeneration when they are separated by round-cells, which ultimately are transformed into fibrous tissue. In these ways an indurated area or scar may be produced which will resemble indurative degeneration.

Purulent myocarditis is produced by septic emboli. Ulcerative endocarditis and pyæmia are oftenest the cause of them. They always produce abscesses. This form of myocarditis is rare. The abscesses are usually small. There may be several of them, or only one.

Symptoms.—There are no distinguishing symptoms. Myocarditis, which grows out of peri- or endo- carditis, produces no other symptoms than those of the primary disease. The scars which may result from it will produce no symptoms, or they may be accompanied by the various ones associated with indurative degeneration. Purulent myocarditis can rarely be diagnosed.

Treatment.—The treatment of myocarditis does not differ from endo- and peri- carditis in the first group of cases or from pyæmia in the other.

DISEASES OF THE ENDOCARDIUM.

CHAPTER XXVI.

ENDOCARDITIS.

Anatomy.—Inflammation of the lining of the heart is a common cardiac affection. Any part of the interior of the heart may be inflamed, but commonly only the valves or their immediate neighborhood is involved. *Acute endocarditis* is almost invariably limited to those portions of the valves which chafe against each other. After birth it rarely affects the right side of the heart, though it commonly does before.

When acutely inflamed the point of attack is at first reddened. The subendothelial tissues are soon infiltrated with serum and round-cells. If the valves are affected, they are thus thickened. The endothelial cells loosen, and are detached. The raw surface is now coated with a thin, opaque film, which may grow, by deposition of fibrin, into a wart-like protuberance as large as a pin-head, or even a bean. These protuberances are grayish, or yellowish, or reddish yellow in color. They are often brittle; break, and form emboli.

Two forms of acute endocarditis exist: The first is known as *septic*, *malignant*, or *ulcerative;* the second as *non-malignant*, *simple*, or *verrucose.* The former is due to septic infection. Micro-organisms abound in the lesions, and produce, when carried elsewhere in emboli, septic infection of distant organs. Septic endocarditis is so uniformly characterized by loss of substance in the valves that it is often named ulcerative. Similar loss of substance may occur, but seldom does, in simple endocarditis. In ulcerative endocarditis, if the valvular vegetations are detached, a loss of substance, with ragged and sharply-cut edges, is laid bare. This ulcerating destruction of the tissues may extend deeply, and may even penetrate a valve. Oftener, where the valve is thus thinned the endothelium upon its opposite surface becomes distended and protrudes. Slowly, it will be stretched out into a sac, with a narrow neck at the point of ulceration. Such a sac is known as a valvular aneurism. It may contain fluid blood or a thrombus. An aneurism may rupture, or even a thin valve that is not aneurismal may.

In simple endocarditis there is oftenest built up on the abraded valvular surface a vegetation, the base of which is composed of round or granulation cells and the upper part of fibrin. Under the microscope the uppermost cells in these vegetations are degenerated and granular. In the deepest layers of fibrin a few atrophied nuclei may still be seen, which have been set free from cells that have disintegrated.

Chronic inflammation may begin as such or grow out of acute inflammation. It is characterized by thickening of the valves, roughening of their surfaces, rigidity, contraction and contortion of them, often degeneration of their deeper tissue, and even calcification. The valves may become partly adherent to one another, or to neighboring parts of the endocardium. The fibrillæ and muscular papillæ may be involved, both in acute and chronic changes. They may be broken, or shortened, or contorted, and thus, also, interfere with the function of the valves. If chronic endocarditis arise by extension of endarteritis, the base of the aortic valves may be chiefly involved instead of the edges, or the upper instead of the lower surface. When the inflammation spreads from the mitral to the aortic valves, or *vice versâ*, the base is also especially apt to be affected.

Instead of lapsing into chronic inflammation or leaving a chronic valvular lesion, acute endocarditis may undergo perfect resolution. This is rare, however. These various changes in the valves modify their function. Their swelling causes rigidity. Chronic thickenings and calcifications make them still more rigid. Verrucosities or chronic roughness will prevent perfect coaptation of the edges of the valves Contraction of them will prevent a perfect closure of the orifice, which they should guard. Their roughness, their slow movement, and other changes will cause unusual eddies and currents in the blood-stream, which produce the modified heart-sounds that are recognized as murmurs.

The imperfect opening or closing of a cardiac orifice by the valves will produce other changes in the heart and circulation. We can best describe these later. (See page 240).

Emboli originating in the heart may cause abscesses, if they are septic; or infarcts, or dropsy, or local anæmia, if they are aseptic.

Causes.—It has been proven that *septic endocarditis* oftenest arises as a complication of puerperal fever or of some other form of sepsis. As the point of infection cannot always be discovered, some cases are described as idiopathic or primary. Several different forms of micro-organisms have been found in the cardiac lesions. It seems quite well established that the disease has not a single specific microbic cause. Old valvular and cardiac lesions are especially the locus of septic endocarditis. After puerperal fever, septic endocarditis oftenest complicates articular rheumatism, infectious exanthemata, diphtheria, typhoid, periostitis, and osteomyelitis.

Simple endocarditis is rarely, if ever, a primary affection. It usually complicates articular rheumatism. It is associated both with the mildest and the severest cases and with subacute and acute. It is said to occur oftenest in those cases in which many joints are simultaneously involved. Gonorrhœal and other rheumatoid affections are rarely accompanied by endocarditis. Chorea is very frequently associated with it. The exanthemata, protracted fevers, nephritis, pleurisy, pneumonia, phthisis pulmonum, and many other diseases may be complicated by it. The tubercle bacillus has been found in endocardial vegetations which developed in a consumptive. Oftenest the valves become irregularly slightly thickened and roughened in phthisis, but not sufficiently modified to cause cardiac symptoms.

Chronic endocarditis frequently grows out of acute attacks, but in many cases its origin is insidious. Senile changes which produce sclerosis in the blood-vessels lead to chronic valvular and endocardial thickenings and induration. Severe and unusual muscular strain also disposes to the disease, as military surgeons have demonstrated among recruits. It often follows nephritis, diabetes, gout, syphilis, chronic lead and alcohol poisoning. Certain irritants in the blood undoubtedly produce it in these affections.

Symptoms.—It is impossible to describe a group of symptoms which characterize all cases of acute septic endocarditis, for no two are precisely alike. Many cases cannot be diagnosed, and many more not without weeks of observation and study. The course of the temperature and many of the symptoms commonly resemble either typhoid or intermittent fever. But the cardiac disease is often unnoticed in the course of a primary affection, such as puerperal fever or some other septicæmia. Those cases which most resemble typhoid have a fever of a continuous type. The patients are apathetic. The tongue is dry and brown; the pulse is quick, soft, and dicrotic; the abdomen tympanitic, and sometimes roseola spots can be found on it. Other cases not only resemble intermittent fever in the course of their temperature, but, as in them, the spleen enlarges; a chill, fever, and sweat recur with uniformity each day, or each second or third day. If the disease runs a long course the fever gradually becomes more continuous and less intermittent, or less regularly so. In both groups of cases there may be physical signs of a cardiac disease, or they may be entirely wanting. The occurrence of embolism is most suggestive of a cardiac lesion. Emboli may cause hæmorrhagic infarcts, or œdema, paralysis, or often abscesses. If they are very minute, and especially if they are in internal organs, they may not manifest themselves. Embolism of the skin and retina can be observed more readily. If the physical signs of a valvular lesion gradually develop, a diagnosis may be made with some positiveness. A gradual, but progressive, loss of flesh and strength takes place. Death is almost inevitable.

Acute non-malignant endocarditis can usually be diagnosed with certainty. If the lesion is not upon the valves, or if it does not interfere with their action, a diagnosis is impossible. Reliance must be placed entirely upon the physical signs, for subjective symptoms may be wanting or indefinite. In rheumatism and chorea the heart should be frequently examined, as valvular lesions may otherwise be overlooked. The temperature may be raised or remain unchanged when the heart is involved. Sometimes oppression is simultaneously felt in the cardiac region; more rarely, pain and tenderness are there. Palpitation or irregularity of the heart first attracts attention to other cases. Syncope and dyspnœa are rare; if they occur, they are usually due to heart-clot or embolism. Oftener no subjective symptom suggests a cardiac lesion, but it is discovered by the development of physical signs. Embolism and its effects will often make positive a diagnosis that otherwise is probable.

If in the course of a disease that is likely to be complicated by endocarditis murmurs arise, we are justified in suspecting its existence and often in affirming it. If diastolic murmurs develop under these circumstances, we are assured that endocarditis has produced the conditions which give rise to the murmur; but, unfortunately, diastolic murmurs are not the commonest. Systolic murmurs may develop in fevers or when there is anæmia, even though no endocarditis exists. Though, as a rule, endocarditis is the cause of systolic murmurs which accompany acute articular rheumatism, I am sure that in several cases I have heard a mitral systolic murmur which was not due to this cause. In these cases the murmurs disappeared entirely when the patient regained strength and his blood its richness. If, however, such murmurs persist, and if, subsequently, changes in the size and shape of the heart develop, which are usual when the valves are permanently modified, we may make a positive diagnosis. If embolism occur, a diagnosis can be made with greater certainty.

While it is impossible always to make a diagnosis of acute endocarditis when it exists, and especially of the malignant form of the disease, a diagnosis is usually possible. The symptoms of most value are those which a physical examination demonstrate or which arise from embolism, and the co-existence of one of the diseases that are commonly regarded as causative. The physical signs which make it possible to determine which valve is chiefly affected are the same that enable us to make the same determination in chronic valvular disease. (See pages 243 to 250.)

Treatment.—The treatment of *ulcerative endocarditis* must be directed to the conservation of strength. General nutrition must be maintained by administering food as it would be to those suffering from continued, intermittent, or septic fever. Milk, gruels, broths, and eggs should constitute the regimen. If the stomach is not retentive, or if its digestive powers are impaired, food is best given frequently, in small amounts, but to others it may be given more generously and less frequently.

Cardiac exhaustion and failure are commonly the immediate cause of death. As the heart grows feeble, it must be spurred to greater efforts by digitalis, strophanthus, convallaria, and similar drugs. When it is extremely feeble, diffusible stimulants, such as ammonium carbonate and camphor, must be relied upon. A great number of antiseptics have been administered in these cases, but unavailingly.

As *non-malignant endocarditis* is a secondary affection, treatment must be addressed to the primary disease.

In the onset of the endocardial inflammation an ice-bag may be constantly applied to the præcordia, or, instead, blisters, followed by fomentations, may be used. They will act as they do when serous sacs are inflamed. The mild chloride of mercury is used as in pleurisy and pericarditis, with the hope that it will modify the exudate and prevent its organization. In subacute and non-chronic cases the iodide of soda or potash is used. Digitalis or analogous remedies must be employed if the heart is unduly weak, irregular, or fast. During convalescence they can be gradually omitted, and bitter tonics and iron can be advantageously used to restore the heart-muscle to a greater degree of nutritial vigor. In these cases, too, nourishment must be carefully administered, so as to maintain strength.

Prognosis.—In malignant endocarditis the prognosis is unfavorable; in non-malignant cases it must be guarded, for, almost without exception, a chronic valvular lesion is produced. If this does not interfere so greatly with the function of the valves that the heart fails by hypertrophy to compensate for it, life may not be shortened. In such cases there is a physiological, though not an anatomical, recovery. But, in many cases, either the lesion is too great to be compensated for or the general vigor of the individual is not sufficient to make hypertrophy possible.

CHAPTER XXVII.

CHRONIC VALVULAR DISEASE.

Nature and Anatomy.—Anatomical deformity of a cardiac valve may exist without disturbing its function. For example, a scar upon a valve may make it abnormal, but it may still open and close perfectly the orifice it guards. No cardiac disease is produced by such a lesion. If a valve is so displaced or deformed that it narrows the orifice it should protect, or leaves it constantly open, its function is not performed, and a more or less extensive change in the heart, and usually in other organs, will be produced.

The valves may be unusually thickened and roughened by inflammation, by degeneration and calcification, or, infrequently, by new growths. They may be contorted by scars; they may be adherent to one another, or to the adjoining wall of the heart; they may be torn; they may be perforated by an ulcer or ruptured aneurism. Their functional activity may be interfered with by rupture, contraction, or degeneration of the muscular papillæ or chordæ tendinæ. A valvular orifice may be dilated and the valves made incompetent though they are not diseased. Some of these lesions may be developed congenitally,—either from imperfect development of the fœtus, or from inflammation or other less frequent and imperfectly understood causes. When lesions are congenital they are usually upon the right side of the heart. In adults they are commonly due to endocarditis; degenerative changes, such as produce arterial atheroma, also produce some of them. A valve is rarely ruptured from strain alone. But usually, as in a case that recently came under my own observation, a severe bodily strain increases the arterial blood-pressure, and causes a rent in the edge of, for example, an aortic valve that has been slightly weakened by degeneration—which may be extensive—in the aorta. Occasionally, when the cardiac cavities dilate, the orifices are also stretched, so that valvular incompetency results. Tumors are very rare within the heart. Sometimes foreign bodies, especially cardiac thrombi, entangled in the chordæ tendinæ and protruding through a cardiac orifice, will produce symptoms that precisely simulate a valvular lesion.

Symptoms.—The general symptoms which accompany a chronic valvular lesion are due to cardiac weakness, and are the same as those accompanying cardiac weakness from other causes. If the heart, by hypertrophy, can compensate for the stenosis or insufficiency which a chronic valvular lesion may cause, general symptoms will not arise. These symptoms are due to a disturbance of the balance between the arterial and venous circulations. The arteries are imperfectly filled; the blood within flows slowly, under diminished pressure. The veins are overfilled, but in them, too, the blood-stream is slow; the pulse, therefore, feels soft, and is small or of medium size. The heart is quick when compensation is not perfect, and often becomes irregular and tumultuous. Physical exertion, mental excitement, or difficult digestion will frequently hasten the heart's action to a distressing degree. Stenosis causes this imperfect vascular balance by making the arterial stream slow, by filling the arteries slowly and imperfectly, and, consequently, making pressure within them low. Behind the point of stenosis the pressure is increased and the veins are overfilled. Insufficiency produces the same results, because of the regurgitation, which also causes overfilling and increased tension in the veins, and imperfect filling and low tension in the arteries.

The imperfect vascular balance leads to passive engorgement of various organs. The lungs and bronchi are commonly thus affected. They may undergo the changes which are characteristic of passive hyperæmia, and that are known as brown induration. Often, when congested, the bronchi become inflamed, and remain persistently so, with varying degrees of severity. Dyspnœa is a common symptom, and may be due to the congestion, to bronchitis, to brown induration, to œdema of the lung, or to pleural dropsy.

The liver may be greatly enlarged from venous hyperæmia. It can then be felt as a smooth body with rounded borders. It may be so large that the lower ribs will be pushed outward. It is usually subject to frequent and very marked variations in size. It is tender, and its distension often causes persistent soreness. If the hyperæmia has lasted long, the liver may gradually contract and its surface may grow rough; it will then become hard. Thus, it is transformed into the condition known as the nutmeg-liver. Often an icteric hue can now be observed in the patient's skin.

The kidneys are also liable to somewhat similar changes. By congestion albuminuria may be caused. The urine becomes moderately diminished in quantity. Its specific gravity is from 1025 to 1035. It is usually turbid, and deeper colored than natural. The amount of albumen present is not great. Hyaline and granular casts can be found in the sediment, but are not numerous. Blood-cells are also often present in small numbers. In this stage of congestion acute inflammation may occur; if prolonged, congestion leads to contraction and cirrhosis. The urine will then increase in amount;

its specific gravity will fall below normal; the albumen will be reduced to a trace, and casts will be rarely found.

Passive hyperæmia of the stomach and intestines leads to slow digestion, constipation, and, finally, often to catarrhal inflammation. These lesions produce corresponding symptoms: indigestion, anorexia, vomiting, flatulence, and constipation may characterize one case; sour stomach, pyrosis, tenderness, or diarrhœa another. Anasarca and dropsy of any of the serous cavities may also result from the imperfect balance of the arterial and venous circulation.

All of these lesions and symptoms do not ordinarily occur in the same case, but they occur in varying combinations. Oftenest a congestive dyspnœa and general anasarca are combined. I have seen cases in which the liver was enormously enlarged by congestion while the lungs were almost unaffected, though respiration was uncomfortable, because the liver impeded the movements of the diaphragm. Indigestion also increased the patient's distress, but there was no anasarca. In other cases the kidneys may be early involved.

Bodily temperature is not changed in these cases, unless inflammation causes it to rise. A slow and imperfectly-maintained circulation leads to slow and imperfect tissue-change. Reparative processes are retarded. Perfect nutrition is not maintained. The disturbances of the pulmonary, gastric, hepatic, and renal functions contribute to malnutrition. The muscles grow small and weak. The blood becomes impoverished, and, therefore, the face is often sallow or anæmic. The patient feels languid and lacks endurance. If dyspnœa is considerable, voluntary exercise may be inhibited. If anasarca is extensive, locomotion may be impossible.

A diagnosis must be based upon the local or cardiac symptoms. The existence of a cardiac murmur, of enlargement of the heart, and cardiac exhaustion are not sufficient to make certain the existence of a chronic valvular disease. I have seen pericarditis and pericardial calcification produce these symptoms, which gradually developed and lasted for several years. I have known muscular degeneration to cause similar symptoms. It is true that under these circumstances the murmur is always systolic, and is usually best heard at the heart's apex. To establish a diagnosis, we must find a cause for a chronic lesion, and we must find, on physical examination, the *combination* of changes which are the result of valvular lesions.

Aortic insufficiency will cause great dilatation of the left ventricle, and, if the lesion is chronic, hypertrophy also; for the ventricle must hold not only its normal quantum of blood, but also what flows back into it through the patent valves during diastole. The other cardiac cavities may not be changed. These lesions cause the præcordia to be prominent. The apex-beat is readily seen and felt. It is diffuse. It is to the left of the nipple, and usually a little lower than is normal. In the supra-sternal notch pulsations can often be seen. The carotid pulse is usually visible, and sometimes a capillary pulse can be demonstrated beneath the finger-nails by the varying breadth of the color-zone with each heart-beat. Pulsation in the retinal arteries can also sometimes be seen. Palpation confirms the diffusion of the apex-beat, and demonstrates its powerful lifting character. This unusual forcefulness is due to hypertrophy. In many cases a diastolic fremitus can be felt at the base of the heart. Percussion demonstrates the cardiac enlargement. The left border of dullness will extend to the left of the nipple, and sometimes even to the anterior axillary line. The area of dullness usually begins a little higher than natural. Rarely, it extends a little to the right of the sternum, in the second intercostal space; this is due to dilatation of the aorta. The right border of the heart remains unchanged, except in rare cases, when it is found farther to the right than is natural, and a substernal beating and accentuation of the pulmonary second sound indicate a dilatation and hypertrophy of the right ventricle. The cause of these changes is usually obscure. It may be due to stretching the mitral orifice, and consequent insufficiency of the mitral valves.

A diastolic murmur characterizes aortic insufficiency. It is generally best heard about the centre of the sternum. This is because the murmur is produced not at the aortic valves or in the aorta, but in the upper part of the left ventricle, where the blood from the auricle and the blood flowing back from the aorta commingle and produce the eddies which cause the murmur. Often the second aortic sound is obliterated by the murmur, but not always. If some of the aortic leaflets can unfold naturally they may produce the second sound. In other cases a second sound may be transmitted from the pulmonary artery. The murmur is usually heard over the pulmonary artery, but not so loud and clear as farther to the right. At the apex there usually is no murmur, and both first and second sounds are normal. Occasionally, a diastolic murmur is faintly heard there, and more rarely a systolic one. The latter is not always significant of aortic stenosis. When it exists a satisfactory explanation of its causation is difficult. It may be due to irregular contraction of the heart-muscles.

In the carotid a systolic murmur is often heard. It is sometimes propagated from the valve; sometimes it is due to irregular vibrations of the vessels, which arise from their excessive tension; or, it is of hæmic origin. At times the signs of aortic insufficiency disappear. This may be due to the stretching of one valvular curtain so that the valvular leakage is stopped, or vegetations may grow so that they can help to close the orifice. Occasionally, an insufficiency is gradually converted into a stenosis.

The pulse is usually full and tense. The artery fills and empties quickly. This is best demonstrated by a sphygmographic tracing in which the ascending and descending lines of the pulse-wave form an acute angle. The rapid emptying of the artery is due to the fact that it both empties forward into the capillaries and backward into the ventricle. On the descending line the diastolic notch is usually shallow, and approaches the respiratory line. In the carotid systolic thrills can sometimes be felt.

If there is *stenosis of the aortic orifice* the blood within the ventricle is under unusual pressure. This causes a slight or moderate stretching, or dilating of the ventricle. But to force the blood through the narrow opening the heart must work hard, and therefore hypertrophies. The aorta fills slowly. The auricles and right ventricle may remain unchanged. The enlargement of the left never attains the great size that it does when there is aortic insufficiency, as it is not dilated by an unusual quantity of blood.

The præcordia is prominent in chests that are plastic. The apex-beat is usually visible, and is generally strong and lifting when felt; but, in some cases, it is unusually weak, and can scarcely be perceived. This is, at least in part, due to the absence of recoil, as the aorta is slowly filled, and, therefore, straightened less than is normal. The apex is depressed and displaced to the left. Palpation often reveals a thrill in the second intercostal space adjacent to the sternum. By percussion the area of cardiac dullness is found to be moderately increased to the left, and rarely to the right. Auscultation demonstrates a systolic murmur which is loudest in the second right intercostal space, adjacent to the sternum. Usually, it can be heard elsewhere, over the heart, and may even obscure the other heart-sounds. It is occasionally heard extensively over the chest and in the back. It can be traced along the aorta, and heard almost always in the carotids. Usually, the second heart-sound is obscured over the aorta and carotids, and often over the pulmonary artery. Generally, it can be heard at the apex. Except over the aorta the normal cardiac sounds may be heard, but oftenest the murmur is transmitted somewhat to all parts of the heart.

The pulse is often slow and, as compared with the apex-beat, is retarded. The pulse is characteristically hard and small. The artery fills and empties slowly. This latter fact is best demonstrated by a sphygmogram, in which the lines are seen to ascend and descend gradually and to form a round-topped wave.

When the mitral valves are affected, changes take place in the heart much more extensively than when the aortic valves are the locus of disease. If the *mitral valves are insufficient*, blood will flow into the left auricle, as usual, by the pulmonary veins, and in the usual amount, but it will also flow in from the left ventricle. Necessarily the auricle must dilate, in order to hold this abnormal quantity of blood. Moreover, as the left auricle contains an unusual amount of blood, the ventricle also must dilate to hold it when it is expelled from the auricle. The overfilling and stretching of the auricle increases the blood-pressure within it, and also in the pulmonary veins. If the mitral leakage is considerable, congestion of the pulmonary capillaries results, and increased blood-pressure is transmitted through them into the pulmonary artery. A heightened blood-pressure in the pulmonary artery leads to slight or moderate dilatation of the right ventricle, and often to very considerable hypertrophy of it. The leakage at the mitral orifice must be compensated by right ventricular hypertrophy, for the weak walls of the auricle are capable of very little hypertrophy, and certainly not of enough to compensate for the results of the usual mitral lesions. Very moderate hypertrophy of the left ventricle is produced by the necessity of propelling a somewhat larger amount of blood than is normal.

From these anatomical changes one can reason to most of the physical signs that are characteristic of the lesion. The præcordia is usually prominent. The apex-beat may be normally located, but generally is immediately beneath the left nipple or to the left of it. The beat is diffuse. The end of the sternum is often raised at each systole. This is significant of hypertrophy of the right ventricle. In the epigastric region throbbing is almost uniformly visible. Occasionally, it can be seen to the right of the lower part of the sternum. This generally happens when the right ventricle is much dilated. Rarely, a systolic impulse has been seen over the pulmonary artery. It occurs when that vessel is distended and lies against the chest-wall. Over the pulmonary vessel a sharp impulse can sometimes be felt, which is synchronous with the closure of the pulmonary semilunars. At the apex a systolic thrill is of frequent occurrence. Occasionally, it can be felt only when the patient leans forward or to the left, or after hurried movements have been made. The area of cardiac dullness is broader than normal. It is often extended to the left, but always noticeably and sometimes greatly to the right. It usually extends to the right of the right sternal border.

Auscultation reveals a systolic murmur which is loudest at the apex. It can sometimes be heard all over the heart. In the rare cases in which the appendix of the left auricle is distended and wrapped around the base of the pulmonary vessel, it may be heard loudest over that vessel; that is, in the second left intercostal space. It can usually be traced to the left of the apex, into the axillary region, and sometimes to the back. It is least frequently plainly heard over the aorta; that is, in the right second intercostal space. A systolic sound is also often audible, and is synchronous with the murmur. It may be transmitted from the tricuspids or produced by the ventricular contractions. Over the pulmonary vessel an accentuated or sharply clicking sound is produced by increased pressure in the pulmonary vessels and by hypertrophy of the right ventricle. The radial pulse is not characteristic, but is especially apt to be irregular if there is imperfect compensation. The sphygmogram is not peculiar, though it usually demonstrates a low arterial pressure.

Mitral insufficiency occurs more frequently than any other chronic valvular lesion. It is very often combined with mitral stenosis, the physical signs of which are wanting. Compensation may be quite perfect, but rarely is as perfect as it may be when the aortic valves are affected. Recoveries have been reported. I have myself observed cases in which mitral systolic murmurs and dilatation of the right heart completely disappeared that had originated in an attack of acute articular rheumatism. But the patients had become anæmic during their rheumatic attack, and I did not feel confident that the murmur was the result of a valvular lesion, for in anæmia and fever dilatation of the right ventricle and systolic apical murmurs may exist. A diagnosis of a mitral lesion is sometimes difficult. An accentuation of the second pulmonary sound is confirmatory of a valvular lesion, as is also a wide distribution of the murmur, especially to the left of the heart.

If there is uncomplicated *stenosis of the mitral valves*, less extensive changes are usually wrought than by insufficiency. Because of the obstruction to the outflowing current from the left auricle, blood-pressure within it is increased. This dilates the auricle somewhat. The increased pressure is, however, transmitted through the pulmonary vessels to the right ventricle, which also dilates, and, in order to compensate for the mitral obstruction, hypertrophies. The left ventricle does not hypertrophy, and may even diminish in size, because the blood within it is under low pressure, and may even be diminished in amount, because of the mitral obstruction.

The præcordia is usually prominent in those whose ribs and cartilages are pliable. Dilatation and hypertrophy of the right ventricle, as in mitral insufficiency, produce a diffuse cardiac impulse, which is visible beneath the sternum or lifting it, and sometimes in the intercostal spaces to the right of it. The apex-beat is seen to the left of the left nipple when the right ventricle is much enlarged.

A fremitus, or thrill, can often be felt at the apex. It is presystolic, or, rarely, diastolic. It is usually confined to the apex. Often it is best, or only, felt with the patient reclining upon his left side or leaning forward, or when physical exertion or mental excitement quickens and makes forceful the heart's movements. A diastolic impulse may be felt over the pulmonary artery, in the second left intercostal space, as in mitral insufficiency. Percussion will demonstrate a right-sided enlargement of the heart. The shape of the area of dullness is similar to that in mitral insufficiency. Murmurs may be wanting, but usually a presystolic, or, rarely, a diastolic, one is heard at the apex. The murmur is often heard less plainly at the end of the sternum, and is least likely to be heard over the aorta. The heart-sounds over the aorta are often feeble. The second sound over the pulmonary is accentuated. At the apex the first sound is clearly heard, the second is sometimes absent. Rarely, a re-duplicated diastolic sound is heard over the aorta and pulmonary artery. This is due to the low blood-tension in the former and high tension in the latter, which causes the valves in the two vessels to close at different times. Systolic murmurs may be heard over the carotids and subclavian. The pulse is usually small and soft; it may be irregular, and often is fast.

A mitral stenosis is rare, except as it is combined with insufficiency. It may gradually develop out of the latter by the growth of verrucosities or by hardening of the valves. The prognosis is less favorable than in mitral insufficiency.

Pulmonary insufficiency and stenosis repeat upon the right side of the heart what aortic insufficiency and stenosis cause on the left. As these lesions are oftenest congenital, præcordial prominence is usually produced in the infant's pliable thoracic wall. The impulse is seen and felt to be diffuse, and especially is it demonstrable at the end of the sternum. Thrills are often felt. If there is insufficiency, they will be diastolic, and will be felt best, or exclusively, in the second left intercostal space. If there is stenosis, they will be systolic, and felt best in the same place, but may be diffused over the entire heart. Percussion demonstrates a right-sided enlargement of the heart. Auscultation reveals murmurs, which are best heard over the pulmonary artery, but may be transmitted toward the end of the sternum. If there is insufficiency, the murmur is rarely heard at the apex, but may be if there is stenosis. The diastolic sound may be absent over the pulmonary artery or feeble; at the other valvular orifices the sounds may be normal. If there is insufficiency, a systolic, as well as diastolic, murmur is sometimes heard in the second left intercostal space, which is due to irregular vascular vibrations, because of excessive tension, not necessarily to a complicating stenosis. Unfortunately, accidental murmurs often simulate pulmonary stenosis. They do not generally produce a fremitus, and are less likely to be accompanied by any considerable change in the right ventricle. Stenosis usually causes cyanosis.

Lesions of the tricuspid valves are rare, and when they occur are usually congenital, though they may be secondary to mitral lesions. Stenosis is so rare that it is of no clinical importance. Its symptoms can be theoretically constructed with ease. *Tricuspid insufficiency* is not uncommonly due to dilatation of the right auriculo-ventricular orifice, though the valves remain normal. This is likely to occur when the right ventricle dilates greatly. Cardiac dullness is increased to the right in this case. A systolic murmur is heard over the tricuspids. I have observed a case in which this murmur was so loud that it obscured all other cardiac sounds, although it was produced by relative insufficiency. It disappeared under treatment, and the primary mitral lesion was revealed. Systolic sounds may be heard over the jugular and cervical veins, and, if the venous valves are insufficient, murmurs may be heard. A venous pulse is especially characteristic of tricuspid insufficiency. It may be seen and felt. It is oftenest detected in the jugular veins and liver; less frequently, in the legs and other distant vessels. The pulsations are not evident, even though there is tricuspid insufficiency, unless the venous valves are incompetent from dilatation of the vessels. The venous pulse is due to stasis in the vessels and the transmission of the pulse from the heart. Rarely, both the auricular and ventricular contractions produce a pulse, but usually it is the latter only that does. When the tricuspids are insufficient the right auricle is distended with blood, dilates, and hypertrophies; the right ventricle dilates a little. The increased tension in the auricle is transmitted to the venæ cavæ and veins. The jugular pulse can often be felt, as well as seen. It can be best distinguished from a pulsation of the underlying artery by compressing the vein in the middle, when it will collapse below the point of compression if there is not a true venous pulse; if there is, it will continue to be seen and felt. A venous pulse is very rarely produced when there is hypertrophy of the right ventricle and no tricuspid insufficiency. Hepatic pulsation is best demonstrated by placing the hands over the liver, in front and behind, when they can be felt to separate with each impulse of the heart. The liver often becomes enormously distended by this passive congestion. There is low pressure in the pulmonary artery. This tends to diminish or prevent pulmonary congestion and consequent dyspnœa; therefore, the liver may be enormously enlarged when there is relative tricuspid insufficiency and a primary mitral lesion, though there is very little dyspnœa.

A combination of valvular lesions occurs very often. They frequently hasten cardiac failure and rarely so counteract one another as to retard it. Stenosis and insufficiency may co-exist at the same valve, or two valves may be simultaneously involved. Combined lesions may be said to be the rule, but are least likely to occur when the arterial orifices are involved. Though combined lesions are so common, they usually cannot be diagnosed during life, for one or the other will so far predominate that the clinical picture will be of a simple lesion. Sometimes the symptoms of a secondary lesion in a combination will supplant the primary. As, for instance, in the following cases: In the first the heart beat tumultuously. There was a loud systolic murmur plainly audible at the end of the sternum and along its right border. It could be heard feebly elsewhere. No other murmur was audible. The heart was considerably enlarged to the right, but not to the left. A diagnosis was made of tricuspid insufficiency, probably due to dilatation of the right ventricle, which, in turn, was caused by a mitral lesion and probably by stenosis. Two days later, when the heart had been slowed and somewhat contracted by digitalis, the murmur first heard was gone, but at the apex a low presystolic murmur was audible. In another case, when the first examination was made, the heart was beating regularly, but rapidly. A loud diastolic aortic murmur was heard, and a low systolic one was suspected, but not plainly audible. Some days later, when the heart was beating slowly, the diastolic murmur was almost gone, but the systolic one was plain. In this case there was evidently a combined insufficiency and stenosis at the aortic orifice, and the murmurs were changed in character by the rapidity of the blood-stream. Such a combination as this last may retard the heart's dilatation and muscular weakness; for the stenosis will prevent a reflux of so large a quantity of blood into the ventricle during diastole as might otherwise take place, and so retards or prevents great ventricular dilatation. During systole the effect of the insufficiency is lessened by the stenosis. Occasionally, the character of the murmurs at different orifices may enable one to diagnose combined lesions, or if both a systolic and diastolic murmur exist we can generally conclude that there is a combination of lesions. An exception to this general statement must be made, since systolic murmurs may be produced by irregular vascular vibrations and may be accidental.

Prognosis.—In general, it may be said that chronic valve-lesions are unfavorable to long life. If compensation is good, life will not be shortened. Sudden death does not often result from them. Simple aortic lesions are least likely to precipitate the symptoms of heart fatigue or exhaustion. If, because of one's social position or occupation, it is possible to avoid all exhausting exertion and still to so live as to preserve general health and vigor, the chances of a long life are good. Unfavorable symptoms are: dyspnœa, palpitation, increasing dilatation of the heart, weakness of the apex-beat, œdema, bronchitis, or other intercurrent disease. Even when œdema is considerable, and arythmia and feeble pulsation characterize the heart's action, it is often possible, by persevering treatment, to remove these symptoms, and by a careful regulation of habits of life to maintain for years sufficient cardiac strength.

Treatment.—We cannot modify the valvular lesion by medicinal treatment, but, by strengthening the heart and by removing all impediments to the circulation, compensation may be established. Physical fatigue must be avoided, and in cases in which compensation is lacking any exertion must be avoided. There are many patients who do not exhibit signs of weak heart except after sudden or violent exertion. Such individuals must be especially careful not to overdo. Indigestion and constipation impede the circulation and help to fatigue the heart. They must be corrected or prevented. Changes of climate and habits of life, which will contribute to one's general vigor, must be encouraged. So much exercise as can be taken without exciting the heart is useful, but in many cases all active exertion is contra-indicated. Under such circumstances, gentle, passive exercise—massage—may be resorted to with good results, as it helps to maintain a better peripheral circulation. When flagging of the heart is evidenced by persisting rapidity of the pulse, and, perhaps, arythmia, digitalis, strophanthus, and their congeners, must be used to strengthen and slow it. They need not be given so often, or in their stead caffeine, strychnia, and other bitters may be used, if the heart is only accelerated by exercise or general movements. Iron is usually indicated, as in other cases of heart-fatigue and weakness. The treatment, in a word, must be the same as has been described for these conditions (page 222).

Complications such as œdema and bronchitis must be met as in other cases of weak heart. When passive congestion causes them, heart tonics like digitalis will often relieve them perfectly. At other times the usual treatment for such complications (see page 225) is also needed.

DISEASES OF CARDIAC INNERVATION.

CHAPTER XXVIII.

TACHYCARDIA, OR NERVOUS PALPITATION.

Symptoms.—By tachycardia is meant a rapid or forceful beating of the heart, which is subjectively recognizable, and is not due to organic heart disease. The heart may beat rapidly and forcefully without being subjectively recognizable, but does not then constitute tachycardia. This often occurs in cases of chronic valvular disease. Tachycardia, when violent, is accompanied by other varying symptoms. Attacks of nervous palpitation may last only a few minutes, a few hours, or even for several days. Between the attacks are intervals of varying length of normal cardiac action.

Sometimes premonitory symptoms give warning of an attack. These vary greatly in character in individual cases. They may consist in a feeling of terror or apprehensiveness, or the heart will apparently stop. Dyspnœa, slight syncope, vertigo, cold sweat, or headache foretell an attack in others. Often the paroxysms occur without premonition.

The attack is characterized by rapid throbbing of the heart and a diffuse and lifting apex-beat. The pulsations are felt by the sufferer and may increase the anxiety, fear, vertigo, or deepen the syncope. In many cases the heart is also irregular. The first sound of the heart, especially at the apex, may be metallic in quality or murmur-like. The changed rapidity of the blood's flow or the abnormal muscular contractions are accountable for these peculiarities. At the apex the diastolic sound is often feeble or almost inaudible. This is probably due to the imperfect filling of the aorta and pulmonary artery by each of the quickly-repeated cardiac contractions. Very rarely, the heart-beats are audible at a little distance from the patient. Usually, the carotids throb violently, and in them a systolic murmur and thrill can frequently be heard and felt. The radial artery is generally full, hard, and quick; rarely, it is small and soft. Two hundred or more beats per minute may be made. Dyspnœa on exertion generally exists while there is palpitation, and sometimes it is experienced even when the sufferer is quiet. It leads him often to seek an upright position, for it is increased by recumbence. Speech may become jerky because of the throbbing against the lungs. Epigastric pain is sometimes complained of. The face often becomes flushed and moist, and rarely is pale or cold. Temporarily, there may be a slight rise of temperature. Frequently, dizziness and faintness are felt.

Attacks commonly terminate suddenly, but palpitation may lessen slowly In many of the cases in which tachycardia frequently recurs there is constantly a quicker beating than is natural, which occasionally increases and produces the subjective symptoms that have been described. Attacks often subside with eructation of gases from the stomach or with vomiting or defecation.

Tachycardia can be readily distinguished from endocardial disease by the absence of a persistent murmur and evidence of changes in the size of the heart's cavities. It is more difficult to distinguish between it and affections of the heart-muscle. It can only be done by carefully studying the history of the case and the exciting causes of the palpitation.

It is impossible to designate the part of the nervous system that is chiefly involved in each case. The nervous disturbance may originate in the brain from hæmorrhage, tumor, softening, etc., or much oftener from joy, fright, or violent mental emotions. I have frequently seen quite persistent attacks excited by fright. In one woman severe attacks recurred almost daily for two years, which originated from excitement produced by lightning that struck near her. Another similar case was excited by witnessing a distressing and fatal railway accident.

Causes.—It may be caused by compression of the pneumogastric or sympathetic nerves, by tumors, or by other lesions. Futile attempts have been made to clinically recognize cases due to paralysis of the heart's inhibiting nerves and those due to irritation of the excito-motors.

Exhaustion of the nervous system, as by mental overwork, excessive venery, loss of blood, excessive lactation, will produce tachycardia. These same conditions predispose to it, as do also anæmia, convalescence from severe disease, or other conditions of general enfeeblement. It is especially apt to occur in those who are hysterical and those with a gouty diathesis.

Reflex attacks are common; indigestion, constipation, uterine, renal, and hepatic colic are frequent excitors of them. The excessive use of tea, coffee, and tobacco is one of the commonest causes. Attacks may occur in childhood, as well as in adult life, and may occur in either sex.

Treatment.—Treatment must vary with the cause of tachycardia in individual cases. If predisposing conditions or causes exist, they must be corrected or removed. Anæmia may have to be treated, or the nervous system rested, and the whole body nourished in order to remove the nervous excitability, which provokes palpitation.

Hysterical palpitation is often difficult to prevent. If fright or some other emotion excited it the avoidance of a repetition of the emotion and mental diversion (such as may be effected by change of scene and surroundings, or of work, or mode of life) will often produce the best results. A class of cases which closely resemble hysteria, though there may be in them no other hysterical manifestations, are those in which tachycardia recurs often, and at times is quite persistent, without discoverable exciting or predisposing cause. They are often helped by the same changes that aid the hysterical. In both relief can sometimes be promptly obtained by means peculiar to each individual. For instance, one person may be able to stop the paroxysms by swallowing bits of ice; another by hot drinks, another by strong coffee, another by drawing a long breath and holding it, another by reclining upon the back, another by pressure upon the abdomen. These various and idiosyncratic modes of relief may change from time to time in the same case.

The use of large amounts of strong tea and tobacco should be forbidden in all cases of palpitation, for they may increase the disposition to the affection, and often are exciting causes of it.

It may be necessary to empty the stomach by emetics when gastric fermentation reflexly excites palpitation. Persistent treatment of the gastric disease will remove the cardiac affection in such cases. If the origin of the reflex irritation is in the uterus or other organ it will require especial treatment.

When tachycardia arises from destruction of nerve-tissue by a structural disease of the central or peripheral cardiac nervous mechanism, no hope of permanent relief can be expected.

Frequently, an ice-bag placed over the heart will check palpitation. A sinapism, similarly placed, often acts with equal efficacy. Of drugs, morphine oftenest gives relief. It must be given in moderate, but not somniferous, doses: a sixteenth, an eighth, or a sixth of a grain is usually efficient. Chloral, bromides, ether, chloroform, hyoscyamus, and belladonna are also used with good results. In the hysterical cases bromides and valerian are very useful.

In anæmic and weak persons strychnia, ergot, and iron are oftenest beneficial. Digitalis may be tried, but frequently proves inefficient. It is most useful in the cases for which strychnia and iron are indicated, and with them should be used persistently for a long time.

The constant electric current has been applied to the neck over the pneumogastric and to the sympathetic with varying results, which have not yet been so analyzed that we can deduce indications for it.

SECTION III.

Diseases of the Kidneys.

FUNCTIONAL INACTIVITY OF THE KIDNEYS.

CHAPTER XXIX.

URÆMIA.

Nature.—Uræmia is secondary to various diseases, but chiefly to renal disorders which diminish the excretion of nitrogenous waste. In its most characteristic form, the onset and course of uræmia is acute, but obscure and variable symptoms may precede or presage an acute attack and characterize its chronic form. The symptoms are of nervous origin. Their precise cause is unknown. It was early supposed that the retention of urea provoked them; but both experiment and clinical observations demonstrated that the quantity of retained urea was not proportioned to the severity of attacks of uræmia, and that even when considerable quantities of it were introduced into the blood the symptoms were not always produced. An excess of carbonate of ammonia in the blood, cerebral anæmia, and œdema are other explanations that are equally untenable. More recently kreatinin and kreatin have been thought to be the poisons which produce uræmia. There is little doubt but that some product of tissue-metamorphosis is the cause. It is not known whether the substance is produced by normal tissue-changes and accumulates in the blood when the kidneys cease to perform their function properly, or whether it results only from pathological tissue-changes. Cholemia and diabetic coma resemble uræmia, and may have a similar origin.

Causes.—Uræmia occurs when urea-producing substances are retained in the blood, or when they are formed in excess. It usually occurs when the urine is much diminished in amount, or, at least, when the nitrogenous elements of the urine are diminished. Mechanical suppression of the urine may cause it. Uræmia rarely occurs when diuresis is profuse. Ammoniacal decomposition of the urine in the bladder or pelvis of the kidney, especially if it is not freely voided, and is, therefore, absorbed, will cause uræmia or symptoms resembling it, that are sometimes called ammonæmia. Of kidney diseases, chronic interstitial nephritis is oftenest productive of uræmia. Acute nephritis is next most likely to cause it, and chronic parenchymatous nephritis is least likely to. It oftener complicates scarlatinal than diphtheritic nephritis. It is rarely associated with waxy kidney or passive congestion, but often with the renal disorders that accompany pregnancy.

Symptoms.—The variable and numerous symptoms which often precede acute uræmia are called prodromata, or are collectively named *chronic uræmia.* Headache is one of the commonest of these. It is usually most severe in the morning, and is often occipital. It may, however, occur at other times, and may be variously located. Gastric disturbances are also common, but are not peculiar. Anorexia only may occur in one case, nausea and vomiting in another, flatulent indigestion in a third, or all these symptoms successively in others. These symptoms must be looked upon with suspicion when they occur in the course of a nephritis. A case under my own observation during this winter illustrated their importance. A young man suffered from chronic parenchymatous nephritis, and had repeated attacks of anorexia and vomiting, which lasted for days. There was no apparent cause for them. They were regarded uræmic symptoms, and were promptly relieved by diaphoretics and diuretics. Unfortunately, he at last refused to submit to sweating, and the gastric symptoms culminated in acute uræmia. Diarrhœa is also a frequent prodromal symptom. Occasionally, intense pruritus suggests a uræmic state. In other cases, or alternating with these symptoms, asthma may occur. The dyspnœic paroxysms resemble perfectly those of asthma of a different origin. (See page 3.) As in a case now under my observation, asthma often alternates with gastric symptoms. Occasionally, insomnia is a prominent and distressing symptom of uræmia. Blindness may develop suddenly, last for a few days, and as suddenly disappear. Its cause is unknown. Deafness or buzzing or ringing in the ears may occur, either coincidently or independently. Numbness of the skin and formication are other variable premonitory symptoms. Delirium or an apparent intoxication may also occur in chronic uræmia. Several times I have observed, during the few hours just preceding an acute attack, hyperæsthesia and pain in the skin resembling a cutaneous rheumatism. In one case there was, for more than two weeks prior to the acute and fatal attack, persistent, and at times almost unbearable, shifting pain in the extremities.

The symptoms of *acute uræmia* are definite in character and easily recognizable. They consist in convulsions and coma. Coma occurs almost invariably, and is usually accompanied by convulsions, but it may occur without them. It generally begins as apathy, which deepens into somnolence, and finally into complete unconsciousness. These attacks may gradually, within a few hours, pass off, or may last for days. The patient generally breathes loudly, at times, if not continuously. Respiration, also, is often irregular, or it may be of the type known as Cheyne-Stokes. Death may occur by a gradual deepening of the coma, but usually results from convulsions, in the course of which respiration ceases.

In other cases convulsions occur suddenly, and unconsciousness only lasts during them. They then resemble epileptic seizures. Sometimes twitchings of the muscles are limited to a small group or to one side of the body. The attacks may occur at intervals of days or weeks, but usually recur frequently. When severe, they may repeat themselves every ten or twenty minutes, or even oftener. Usually, coma exists between the paroxysms. If the disease take a favorable course, convulsions will occur at longer intervals, or will finally cease; or, if a fatal course, they increase in frequency, severity, and duration. When chronic nephritis disposes a patient to uræmia, he will usually suffer from the chronic form of the malady, even if he recover from the acute. During convulsions the bodily temperature is often elevated, but may be even subnormal. The pulse is usually quick, soft, and small. It often becomes irregular. Just before an acute attack it is sometimes abnormally slow. During uræmic attacks the pupils are usually normal or large. The sweat and sputa of uræmic patients often exhale a urinous odor. Urea has been found in them, and sometimes crystallizes on the skin or in the hair of the head or face. The urine is usually diminished just prior to and during an attack, and increases when it subsides. In these rarer cases in which it increases before the attacks, its solid constituents diminish greatly. During prolonged coma there is generally incontinence of urine and fæces.

Diagnosis.—A diagnosis of acute uræmia is usually easily made directly from the assemblage of characteristic symptoms. It must sometimes be differentiated from other diseases, and especially from epilepsy, apoplexy, and alcoholism. The convulsive attacks are very like those of epilepsy. The convulsive movements are seldom unilateral, as they usually are in epilepsy. In the latter disease the history of prior attacks, the occurrence of an initial cry, and the absence of albumen are distinctive symptoms. Epileptics become deeply somnolent after their attacks, but not truly comatose. Hysterical convulsions will seldom be mistaken for uræmic, because the sufferer rarely becomes truly unconscious; the convulsive movements are oftener irregular, affect a single extremity at a time, or, when general, usually cause opisthotonos. Commonly after an attack a large amount of non-albuminous, limpid urine is voided. Cerebral apoplexy is sometimes accompanied by convulsions. They are associated with paralysis. In epileptic and hysterical convulsions there is rarely a rise in temperature, which often occurs in uræmia.

The coma of uræmia is differentiated from apoplexy by coincident paralysis in the latter; from opium poisoning, by its normal temperature and the contracted pupil. It is distinguished with more difficulty from alcoholism, as the two conditions are occasionally associated. A history of alcoholism, the odor of liquor in the breath, and no rise of temperature, are indicative of alcoholic coma.

Treatment.—The symptoms of chronic uræmia always indicate that the elimination of nitrogenous matter by the normal channels must be stimulated. In all cases in which uræmia threatens, or may possibly occur, a diminution in the quantity of urine, and especially if its specific gravity is low, must be regarded with apprehension. The kidneys must at once be stimulated to greater activity.

Rest is essential, in order that the amount of muscular waste may be lessened. An abundance of fresh air is also needful, for it insures as good an oxidation of the blood as is possible, and therefore as perfect metabolism of the tissues as is possible. A prescribed diet is also of great importance. Albuminous or richly nitrogenous foods are contra-indicated, for they will increase the danger of surcharging the blood with the class of ingredients which produce uræmia. When it is necessary to exercise the utmost caution to avoid uræmia, a patient may, for a few days, be fed only water-gruels made of barley-meal or arrowroot. Such a diet is not sufficiently nutritious to be maintained long. In three or four days other farinaceous articles must be employed, such as rice, potatoes, and turnips. When the uræmic symptoms disappear, milk and milk-gruels may be administered. A complete mixed diet should be resumed very gradually, and with much caution.

No specific remedy is known by which the uræmic poison can be counteracted. Purdy ("Bright's Disease and Kidney Affections") recommends, as almost a specific in chronic cases, the subcarbonate of iron. It should be given in doses of 1 to 2 grammes (20 to 30 grains) every two to four hours. It is said to rapidly relieve headache, nausea, and other symptoms. My own experiments with the drug have afforded conflicting results. The mode of action of this remedy cannot be explained.

Copious draughts of pure water are also helpful, as they will dilute the blood, thus making it somewhat less toxic, and they will promote more copious excretions from the various organs of elimination. The water should be as free from mineral products in solution as possible, that its dissolving powers may be as great as possible.

The salicylates and benzoates have also been frequently used. They unite with uric acid, and make it more soluble and, therefore, more easily eliminated. It has not been proven, however, that they make other imperfectly oxidized products of tissue-change more soluble. Their utility in uræmia is, therefore, *à priori*, doubtful, and it has not been established by their trial. They are mildly diaphoretic, and in this way will do good. In my hands the salicylate of ammonium is most certain to produce diaphoresis, and will produce it more copiously than any other of this class of remedies. It is, therefore, to be preferred in uræmia. In chronic cases it can be best administered in a hot drink, such as hot lemonade. The patient should at the same time be well covered with clothing, that sweating may be encouraged.

In both acute and chronic cases reliance must chiefly be placed upon drugs that promote elimination from the skin, the intestines, and the kidneys. Jaborandi, or, better, its active principle, pilocarpine, may be administered to produce sweating. Pilocarpine will provoke copious diaphoresis very quickly if it is administered subcutaneously in doses of 6 to 8 milligrammes ($\frac{1}{8}$ to $\frac{1}{10}$ grain). These drugs, although so efficacious as diaphoretics, are contra-indicated in individual cases in which the heart's action is feeble, for they enfeeble it still more. Occasionally, their administration has been known to precipitate pulmonary œdema. It is safer to rely upon hot-air baths to provoke sweating. Air which is heated by a lamp can be readily conducted under bed-clothes by a bent stove-pipe. The patient, reclining upon a bed, should be thickly covered with blankets. The desired effect will be attained most promptly and most perfectly if a hot drink is administered when the hot-air bath is begun. Sweating may be advantageously provoked daily, or even oftener in extreme cases. I have seen patients who were comatose, and occasionally convulsed, brought to a state of consciousness by this means. In two fatal cases life was prolonged for several days and periods of consciousness were established, though permanent consciousness could not be restored. If a patient is very œdematous and is suffering from chronic uræmia sweating must be resorted to with care, for it has been known to provoke acute uræmia. This is due to the sudden re-absorption by the blood of œdematous fluid which held in solution uræmic poison. The re-absorption was provoked by the elimination of the blood's water through the sweat-glands. Under such circumstances only moderate sweating should be produced, or the œdematous fluid should be first withdrawn through incisions at the ankles. (See page 293.)

While elimination of the uræmic poison can be most certainly and most rapidly and perfectly accomplished by the skin, it can be helped by catharsis and diuresis. The cathartics which will accomplish the most good produce copious liquid stools. If frequently employed, they may provoke a catarrhal inflammation of the bowels or cause great weakness. They are best employed occasionally as an adjuvant to diaphoresis, and in persons who are fairly robust. Salts may be administered in a concentrated solution with excellent effect. For instance, if 30 grammes (1 ounce) of Rochelle salts or magnesia sulphate, dissolved in 60 cubic centimetres (2 ounces) of water, is administered when a patient first awakens after a night's sleep, and if no more water or fluids are drunk for some time, several very copious liquid passages will be produced. Elaterium is deservedly a favorite hydragogue cathartic. It may be given in doses of 1 centigramme ($\frac{1}{6}$ grain). The compound extract of colocynth may be used, in doses of from 2 decigrammes to 1 gramme (3 to 15 grains). Croton-oil is also efficacious in its usual dose of 1 minim.

Diuresis should always be attempted, but disappointment often results from the attempt. In mild chronic cases sufficient elimination can generally be thus accomplished to relieve the symptoms, but in cases of greater intensity diuretics do not suffice. They are frequently useful when, by diaphoresis or catharsis, the symptoms have been removed, and it is desired to steadily maintain a moderate increase of elimination of the uræmic poison, that it may not again accumulate in toxic quantities. The diuretics oftenest used are digitalis, strophanthus, acetate and citrate of potassium, ammonium, or lithium. These are often well given in combination. They must be adapted to individual cases, for they may be contraindicated in certain renal diseases, as will be explained later. Digitalis and strophanthus produce diuresis chiefly by supplying to the kidney more blood under higher pressure, so that filtration of the blood's water is accelerated. If the blood is cyanotic because of the feeble action of the heart, and therefore the renal as well as all other tissues are acting imperfectly on account of insufficient nourishment, digitalis will help diuresis by strengthening the heart, and thus promoting a better general circulation. Oxygenation, tissue-nutrition, and action are, therefore, improved.

Potassium, acetate, citrate, and carbonate increase the fluid of the urine, but also, to a marked extent, the elimination of uric acid and extractives like kreatin and kreatinin. The citrate of potassium can be administered in the most agreeable form. Two cubic centimetres (a ½ drachm) of it and the carbonate can be mixed with a small glass of lemonade and drunk while effervescing. The acetate is equally efficacious, but not so agreeable to take. The value of water as a diuretic must not be forgotten. It can be administered freely in all uræmic cases.

Life can sometimes be saved by subduing uræmic convulsions by complete anæsthesia with chloroform, ether, chloral, or morphia. Many cases end fatally because the convulsions stop respiration, or impede and weaken the circulation. By preventing the convulsions such a result may be avoided, and time may be gained in which by diaphoresis and catharsis the uræmic poison can be eliminated. It is usually best to check the convulsion by inhalations of chloroform, ether, or by hypodermatics of morphia. After convulsive movements cease, and before new ones are excited, a large dose of chloral, 2 grammes (30 grains), should be administered to prevent their recurrence. If this is successfully accomplished, as the effect of the chloral subsides a smaller dose must be given, and the drug repeated in successively smaller doses until the uræmic poison is eliminated and its re-accumulation prevented. Opiates retard the elimination of nitrogenous waste, and, therefore, in renal diseases, cannot be steadily used with safety. But they can be used temporarily to subdue convulsions when diaphoretics, cathartics, and diuretics are simultaneously employed.

Individual cases may require the use of cardiac stimulants, such as ammonia or camphor; or tonics, such as digitalis. Other complications in particular cases may also require special treatment.

Prognosis. — Uræmia is a dangerous condition. Chronic uræmia is usually readily amenable to treatment. It is to be feared, as it indicates the possibility of an acute attack. Acute uræmia is very fatal. Braun asserts that 30 per cent. of cases of puerperal convulsions are fatal. Uræmia which accompanies acute nephritis is less frequently fatal than that which accompanies chronic renal inflammation. The immediate prognosis of uræmic coma without convulsions is more favorable than with them, for there is more opportunity of effecting an elimination of the uræmic poison. But such coma oftenest occurs in the most chronic form of renal disease, and, therefore, the ultimate prognosis is not good.

DISEASES OF RENAL CIRCULATION.

CHAPTER XXX.

PASSIVE CONGESTION OF THE KIDNEYS.

Causes.—This lesion of the kidney may be produced whenever the normal balance between venous and arterial blood is so disturbed that the veins are overfilled. Such a disturbance results from cardiac disease. It is commonly associated with chronic valvular disease, but may be with degeneration or other disease of the heart's walls or of the pericardium, which makes the organ weak. Passive congestion of the kidneys may also be the result of obstruction in the venous circulation in the lungs or between the kidneys and heart. Of lung diseases, emphysema, chronic bronchitis, interstitial pneumonia, [illegible] phthisis and chronic pleural diseases may [illegible] passive renal congestion. Cirrhosis of the liver may cause sufficient obstruction in the venous current from other abdominal organs to the heart [illegible] engorgement of the kidneys. Much more rarely abdominal tumors and the pregnant womb compress the inferior vena cava, or veins from the kidneys to it, and cause renal congestion. Thrombosis of the renal veins or vena cava is a very rare cause of the lesion.

Anatomy.—The anatomical changes in the kidney, when it is passively engorged, vary somewhat with the rapidity of their development. As usually observed, the kidney is enlarged, especially by congestion of the cortex. If the congestion develop very slowly the organ may be little or not at all swollen, and, if it is rapidly developed to a considerable degree, it may be greatly enlarged. The color is characteristically dark brown or purplish. The stellate veins are very plainly visible because distended. Whenever the congestion is considerable, hæmorrhages, usually minute, are observable; but if the lesion develop very slowly the organ will not be so dark, and no hæmorrhages will be discoverable. If it is very chronic the kidney becomes paler, smaller, firmer, the surface roughened, and the capsule adherent in places. Ordinarily, the kidneys are firm, elastic, smooth, and the capsule can be readily stripped from the cortex. When a section is made through the organs, the cut surface is characteristically purplish in color, but the glomeruli can be seen with unusual distinctness, as red dots which are arranged in rows. The medulla has a striated appearance, which is plainest where congestion is the deepest. The striation is due to the venous distension. In the more chronic cases, when the kidney's surface is paler, the cut surface is also.

Under the microscope the veins and capillaries are seen to be stretched by the blood-cells. Often small hæmorrhages into the glomeruli or tubules can be found, or the site of former hæmorrhages can be identified by the brownish pigment-granules that may be seen in the tubules, or epithelial or connective-tissue cells. More rarely black melanin casts are observable, which have been formed of the modified blood-pigment. The glomeruli are sometimes a little enlarged. In very chronic cases, when the kidneys have contracted, patches of fibrous tissue can be found here and there, especially just beneath and usually adherent to the capsules. In these areas the glomeruli are often contracted, fibrous, or homogeneous and hyaline. The tubules are obliterated or compressed.

The epithelial cells may appear nearly normal, but they are often in places, and sometimes extensively, large and granular, or contain fat-droplets, or are partly disintegrated. When enlarged they will nearly fill the tubules. In the latter hyaline casts are occasionally observable, or a few red corpuscles, and, less frequently, epithelial cells. The walls of the blood-vessels are usually thickened.

The fibrous patches are probably due to interstitial inflammation which arises about the blood-vessels or spreads from venous and arterial walls. It causes compression of the tubules and consequent atrophy and destruction of them.

Symptoms.—The symptoms of passive engorgement of the kidneys are superimposed upon, and associated with, those of a primary disease. For instance, the usual features of mitral-valve disease of the heart may be accompanied with the evidences of renal engorgement. In such cases usually the pulse and heart are weak, and there is general œdema and dyspnœa. The characteristic signs of the lesion must be sought for in the urine. Generally the heart-lesion or other primary affection and its symptoms exist for weeks, or months, or even years, before the kidneys are seriously involved.

The urine is usually diminished in amount. It is strongly acid in reaction. Its specific gravity is from 1025 to 1035. It is deeper colored than is normal. A relative and sometimes an absolute excess of urates and uric acid must be expected. Uric-acid crystals are frequently deposited in the urine when it stands. If, as is usual, a urinary sediment exist, it is composed of these chemical substances. Often a few blood-corpuscles can be found in it, and a few hyaline or, rarely, granular or blood casts. Epithelial cells from the kidney are also rarely observed. Oil droplets or granules must not be expected. The urine usually contains a small amount of albumen,—a fifth of 1 per cent. or less.

Uræmic symptoms are rare, for the comparatively normal condition of the renal epithelium enables it to eliminate nitrogenous matters. Renal congestion is frequently transformed into nephritis, for it predisposes to inflammation of the organ.

Death commonly results from the primary disease, and is not produced by the renal lesion. The renal complication is significant of the gravity of the primary trouble. Renal engorgement may occur repeatedly in the same case during a period of several years, or, after developing, may persist. The duration of the diseases which produce this renal lesion is uncertain and very variable. The diseases are always, sooner or later, fatal. Whenever a cause for passive engorgement of viscera exists, the urine should be examined. If there is evidence of a heart lesion and engorgement of the lungs or liver, and the urine presents the features just described, a direct and positive diagnosis can be made. It must be differentiated from acute and chronic nephritis. (See page 288.)

Treatment.—Passive renal congestion requires no peculiar treatment. Its cause must be removed. If, as is oftenest the case, a feeble heart cause it, the latter must be relieved of unnecessary work by bodily rest. In some cases, after a few days spent in bed, the symptoms of renal congestion will disappear. Strophanthus, digitalis, and similar heart tonics are indicated. The treatment must be that which we have already described as essential for weak heart. (See pages 206, 222.)

Nephritis must be guarded against. Cold, damp, and changeable weather should be avoided. The body should be protected from sudden atmospheric changes by clothing it with woolen under-garments.

If the quantity of albumen in the urine is for this disease considerable and the quantity of urine small, foods, beverages, and drugs that irritate the kidneys should be avoided. Under such circumstances, albumens should be taken sparingly; alcoholics should not be used; and cubebs, turpentine, and other drugs that are renal irritants are contra-indicated. As a rule, the diet should be generous and highly nutritious, in order to maintain as good cardiac vigor and general health as is possible.

RENAL INFLAMMATIONS.

CHAPTER XXXI.

ACUTE NEPHRITIS.

Causes.—Acute nephritis is especially apt to complicate the eruptive fevers, infectious and septic diseases. Of these maladies, scarlet fever and diphtheria are oftenest accompanied by nephritis. It has been supposed that in such cases micro-organisms which caused the primary ailment were also the cause of the nephritis. This has not been proven. It seems quite as probable that irritating chemicals which are formed under conditions of malnutrition that accompany the primary disease are the cause of the renal inflammation. It must be acknowledged that many observers have found micro-organisms of different kinds in diseased kidneys. Their presence where the inflammation was most intense suggests their causative relation to it. But more numerous observations are needed to confirm the scattered ones already made. The kidneys appear capable of eliminating some micro-organisms in small numbers, but if they are required to attempt the elimination of many the interstitial tissues are found filled with them, or obstructing clumps of them are seen in the tubules. If such collections take place in man, as may occur in animals experimented upon, they may sometimes provoke an inflammation. Such substances as cantharides, turpentine, salicylic and carbolic acids, when ingested, are eliminated by the kidneys, and will cause nephritis when absorbed into the blood in large amounts. Cases of acute nephritis, the result of turpentine poisoning, are not very uncommon. I have observed them oftenest during the summer, in painters using turpentine in close and overheated rooms. Extensive burns of the skin have produced acute nephritis, but in what way is undecided. It may be that the kidneys are overworked, as they attempt to vicariously perform the eliminative functions of the skin, or that they are irritated by substances which should be eliminated through the skin, and which, because of injury to the latter, accumulate in the blood. The causation of many cases cannot be satisfactorily explained.

They often occur after sudden exposure to cold and dampness. Acute nephritis, therefore, is observed most frequently in seasons and in climates that are characterized by changeability of temperature, with moisture. It has been claimed that at times nephritis is epidemic. The cases that have been collated are, however, too few to prove its epidemicity.

Acute nephritis may occur at any age, but is oftenest observed in early adult life. Men are oftener subject to it than women, if we do not take into consideration puerperal nephritis.

Albuminuria and other urinary changes indicative of renal disease are of common occurrence in the course of continued fevers like typhoid. The evidence of renal trouble is usually slight, reaches its maximum when the fever is most severe, and subsides with it. These are cases of acute degeneration or cloudy swelling, rather than of true nephritis. Occasionally, some nephritis accompanies the degeneration.

Anatomy.—The pathological changes characterizing acute nephritis, as might be expected from the varying etiology, are not uniform. An affected kidney may remain normal in size, but usually it is more or less enlarged. In some cases it is greatly swollen, chiefly because of an interstitial serous exudate. The surface of the kidney is smooth, and the capsule will strip from the cortex with ease. The color of the surface varies from red to pearly yellow, or is mottled red and yellow. When the organs are enlarged the increased size is due to thickening of the cortex, for the pyramidal part of the organ remains unchanged in size. The red kidneys are usually of a dark hue, and so much congested that blood will drip from their cut surface. The glomeruli are often prominent as red dots. The mottled form contains less blood. The cortex in the pale form is in places—and sometimes diffusely—whiter than is normal, although the pyramids are dark-red, and often appear striated. The lighter color of the cortex is due to fatty degeneration.

The microscope demonstrates that the focus of the lesion varies in individual cases. Sometimes the glomeruli and their neighborhood only seem inflamed; sometimes an interstitial serous exudation, with some degeneration of the connective-tissue cells, is the important change; in other cases the epithelium of the tubules is chiefly affected. In the cases of acute albuminuria in which there is the least renal change the lesion is a degeneration of the epithelium of the tubules. To the unaided eye the kidneys appear unchanged, or only a trifle large and pale. Under the microscope the epithelial cells are seen to be swollen, so that they nearly close the tubules; they are more granular than natural, and may have no nucleus, or in them it is obscured. The interstitial tissues, glomeruli, and blood-vessels may remain unchanged. Such lesions are oftenest observed in cases of typhoid fever, diphtheria, or other fevers of an asthenic type. They are rather due to acute degeneration than to inflammation. The condition is denominated *cloudy swelling.* Fatty degeneration may grow out of it, or it may be associated with true inflammation. *Fatty degeneration* may occur acutely, for it need not develop from cloudy swelling. It is oftenest the result of infectious diseases, or anæmia, or phosphorus poisoning. When fatty degeneration exists the epithelium of the tubules and glomeruli are not only granular, but oil-droplets are demonstrable in them. Cells that are fattily degenerated oftener disintegrate than those cloudily swollen. If inflammation as well as degeneration exist, inflammatory exudates will also be observed. Degenerative changes may precede inflammatory ones, but quite as often follow them. When inflammation exists some degeneration can also be demonstrated. In certain cases the kidneys are greatly enlarged and softened. In them the interstitial lymph-spaces are usually distended with a serous exudate. A few migrated round-cells may be found about the vessels, but the inflammatory exudate is chiefly serous. In specimens preserved in alcohol small clots of albumen can be seen within the glomeruli or in the lymph-spaces. The renal epithelium is usually granular and degenerated.

In other cases a cellular exudate can be found about the interlobular vessels. The tubular epithelium is usually partly degenerated, and often extensively cast off. The swollen cells or their detritus fill some tubules; the calibre of others is increased by the loss of epithelium.

In other cases, oftenest in those which follow scarlatina and poisoning by terebinthines, the glomeruli are chiefly involved. The capsule of Bowman is usually thickened, the nuclei in the capillary tuft are increased, and the tuft is enlarged. Round-cell infiltration can be seen about the glomeruli. The tuft of capillaries, instead of being changed, as just described, may become homogeneous and vitreous from hyaline degeneration. Small hæmorrhages are frequent in and about the glomeruli. In all forms of acute nephritis minute hæmorrhages are common, and they may occur anywhere in the cortex.

We often find glomeruli, tubules, and interstitial tissues all affected, but in varying degrees. In some cases epithelial desquamation from the tubules is very moderate, in others very extensive. So the amount of hæmorrhage varies, but it is present to some extent in almost every case of acute nephritis. Some of the blood-corpuscles find their way into the tubules and are eliminated with the urine. It must not be supposed that all the glomeruli are equally, or even at all, affected. Usually, some remain perfectly normal, and continue to imperfectly perform the functions of the whole organ. When the kidney is congested the vessels, especially the interlobular ones, are distended with blood. The medulla of the kidney is usually little modified. Sometimes, when degeneration is extensive or congestion very great, it may be affected slightly, as the cortex is extensively.

Acute degeneration and acute inflammation prevent the physiological action of the kidney. These lesions cause, almost without exception, albuminuria. What is much more important, the separation of waste nitrogenous matter from the blood, and its elimination through the urinary channels, is lessened or prevented. Therefore, the liability to surcharge the blood with these products of waste, and the danger of uræmia because of them, is considerable.

Diuresis is interfered with by the various changes which may occur in the glomeruli, and also by obstructions in the tubules, which may be formed of desquamated cells or their detritus or of hyaline casts.

Symptoms.—The disease may develop insidiously, and not be suspected until it produces striking symptoms, such as œdema. The milder cases, especially those that complicate fevers, may not be suspected unless the urine is frequently examined. A diagnosis can be made from the changes observed in the urine, but in all moderately severe cases there are other symptoms that are characteristic. In the cases which develop insidiously, œdema is usually the first symptom to attract attention, though some patients notice changes in the frequency of urination or in the appearance of the urine. In a smaller proportion of cases a chill announces the onset of the attack. This is followed by a rise of temperature which will last a few days. Headache usually accompanies the onset. Sometimes, a dull aching is felt in the small of the back, but oftener backache is absent. The urine is observed to be scant, turbid, and deeply colored. The skin rapidly grows pale, and, as a rule, general œdema is noticeable after the first two or three days. These constitute the most striking symptoms of the sharply-acute cases. Very many, however, run an apyretic course. General œdema exists, as a rule, but in mild cases it may be absent, and is usually in those cases that are acute degenerations rather than inflammations. The œdema is generally first observed about the ankles, or simultaneously about the feet and in the eyelids. At times patients become rapidly and excessively œdematous, so that feet, hands, face, and body are greatly swollen. The eyelids rarely are so filled with œdematous fluid that they cannot be opened, and are distended beyond the forehead. Usually dependent parts only are much œdematous. The feet, the penis, scrotum, and, less extensively, the hands, are oftenest affected.

We must search for the pathognomonic symptoms in the urine. It is diminished in amount, and in extreme cases is almost suppressed. It will measure from 125 to 500 cubic centimetres (4 to 16 ounces) in twenty-four hours. It is reddish or brownish red and turbid. Sometimes it is bloody. As a rule, blood-corpuscles can be found in it in varying numbers, and often are very abundant. Its specific gravity is increased to from 1030 to 1040. It is acid in reaction. Albumen is moderately, and, may be, very abundant in it. There is usually from 0.5 to 1 per cent. of it, and there may be as much as 3 per cent. In individual samples of urine there may be an excess of urea, but during twenty-four hours its amount is from 25 to 50 per cent. less than normal. The other solids are also diminished. If allowed to stand, a sediment is deposited in the urine. In some cases it is composed chiefly of red blood-cells, in others of epithelium, but in most cases of both these elements, together with casts and urates. The blood-cells often are attached to casts, and sometimes cover them, but are most abundant as free cells. Clots do not form in the urine even when hæmorrhage is copious, providing it is from the kidney. Epithelial cells are usually observed in large numbers, and are recognized because they are small, cuboidal, or rudely rounded, and contain large nuclei. If the disease has lasted long, fat-drops can often be seen in them. Earlier, they are granular. While they are most abundant as free cells, they frequently are adherent to hyaline casts, and more rarely are adherent to one another, forming hollow tubes. Casts of all kinds are observable in acute nephritis. Usually, particular kinds predominate in individual cases. Blood and epithelial casts, such as have just been described, are often seen. Granular and hyaline ones are also common. Long, narrow casts are usually formed in the early stage of the disease, and short, broad ones later. Besides these formed elements, granular matter, which is the detritus of disintegrated cells, is usually abundant. Crystals of uric acid, urates, and oxalate of lime often compose part of the sediment. Oil-drops are rarely seen in the urine of acute cases. Micturition is frequent and accompanied by painful straining.

The anatomical changes in the kidney readily account for the abnormal character of the urine. Hæmaturia is due partly to hæmorrhages in the kidney, and partly to the abnormal permeability of the renal vessels, especially in the glomeruli. The changes in the glomerular epithelium and capillaries permit the escape of albumen and lessen diuresis. The obstruction to the tubules by casts, epithelium, and its detritus still more interfere with a copious flow of urine. The degeneration and desquamation of the renal epithelium account for the diminished excretion of urea and other urinary solids.

The loss of blood by the kidneys leads to anæmia, which in turn produces the waxy whiteness of the skin characteristic of these cases. Not only does the blood lose many of its cells, but it is poor in albumen, and its specific gravity is lowered. Urea, uric acid, and other excrementitious matters accumulate in it. The heart is rarely hypertrophied in acute nephritis, but it is sometimes acutely dilated. Pericarditis is an occasional complication. The blood tension in the arteries is usually increased. The pulse-rate varies with bodily temperature, or it may be quickened if the heart is dilated and weak. Bronchitis is a frequent complication. Pleurisy occurs often and tends to become purulent. Pneumonia is an occasional complication and an especially fatal one. Pleural and pericardial dropsy, as well as ascites, are accompaniments of general dropsy, and may be the immediate cause of death. Uræmic asthma is another respiratory complication that is sometimes observed.

Appetite is usually much diminished or almost wanting. Nausea and vomiting occur occasionally. At first they probably are of reflex origin, and due to renal congestion and irritation. Later, they may be uræmic symptoms. The bowels are usually constipated, but occasionally there is diarrhœa, which may be due to intestinal œdema or to a vicarious elimination of abnormal matters from the blood. Uræmia is especially apt to occur in this disease. Transient uræmic amaurosis occurs occasionally. Retinal hæmorrhage, nose-bleed, or other hæmorrhages than hæmaturia are not common. Life is chiefly endangered by uræmia and various complications, such as pneumonia, pleurisy, pericarditis, dropsy of the serous sacs within the thorax, or œdema of the lungs or of the glottis.

Relapses may occur repeatedly after convalescence is apparently established. In this way the course of individual cases may be much protracted. The duration of mild cases is from one to two weeks, but other cases may be protracted from four to twelve weeks. A small proportion of acute cases become chronic.

Uræmia develops from failure to properly eliminate waste matter by the kidneys. It must be suspected whenever the urine is greatly diminished in amount, and especially when the total nitrogenous waste for twenty-four hours is greatly lessened. These are signs of gravity. The amount of albumen voided is not significant of the severity of the disease or its dangerousness. When improvement begins, the urine grows more abundant, hæmaturia ceases, the albumen lessens, casts are less numerous, shorter, and more broken. If there has been much dropsy, and if it is being absorbed during convalescence, the urine will become unusually copious, and its specific gravity may be abnormally lowered.

Diagnosis.—A diagnosis is usually not difficult, and may be made from a urinalysis. In mild cases there may be no signs of the disease, other than those that are discoverable in the urine. It may otherwise be wholly masked by a primary disease. But in some cases the general symptoms are quite as pronounced as the urinary ones.

A direct diagnosis can be made positively when there is associated with the characteristic urinary changes general œdema, rapidly developed anæmia, and a history of sudden onset and of no previous attacks of a similar kind. Acute nephritis must sometimes be differentiated from chronic venous hyperæmia and acute exacerbations of chronic parenchymatous nephritis. From the former it can be distinguished by the larger amount of albumen and a greater number of red blood-cells in the urine, and by the development of œdema, which usually is as quickly observed about the eyelids as in the feet. When passive congestion of the kidneys exists, there must also be one of its essential causes. When in the course of chronic parenchymatous nephritis acute exacerbations occur, there are superimposed upon the symptoms of the chronic malady those of the acute. The urine may be almost undistinguishable from that of acute nephritis. Usually, however, oil-drops and fatty epithelial cells or fatty casts are abundant. A diagnosis must, however, be chiefly based on the history of prior attacks of the same kind, or at least of œdema and other symptoms of chronic nephritis.

Treatment.—The indications for treatment are, first, to give the kidneys, as far as possible, a rest; second, to limit congestion; third, to remove obstructions in the renal tubules; fourth, to guard against or mitigate complications or sequelæ.

To meet the first indications, those who suffer from acute nephritis should remain in bed, for exercise will increase nitrogenous waste, and therefore its elimination, which means renal work. They should live upon a non-nitrogenous diet. This last requirement must be rigidly adhered to only in the most severe cases, or when uræmia most threatens. But in all cases the diet must be so modified that it will contain only a small amount of nitrogenous matter. Water-gruels made of arrowroot or barley-meal may afford a modicum of nourishment for two or three days, until the height of the attack is passed. Fine wheat-flour, rice, potatoes, and turnips are additional foôds that contain very small amounts of nitrogen or vegetable albumen. Apples and grapes are also somewhat nutritious, and may be grateful as condiments. These latter articles can be employed in the less severe cases, or after improvement is established. It is best to administer nourishment in moderate amounts and often, so that it will not be too long undigested, as otherwise it may irritate the alimentary canal by feeding gastric or intestinal fermentation. In the periods of greatest severity, or when uræmia most threatens, even milk should not be used, but during convalescence it is particularly wholesome, for it is a diuretic, very nutritious, and easily digested. It may with advantage, at this time, constitute the chief element of diet. When a normal nitrogenous elimination by the kidneys has been re-established, other albuminous food can be tried with caution. The fact that gastric juice and probably other digestive secretions are lessened in nephritis makes it necessary to guard against filling the stomach with food, but enough must be given to maintain nutrition.

A patient should remain in bed until œdema has wholly disappeared and urea is voided in normal quantities. Exercise should not be continued if by it the elimination of urine and nitrogenous matter is lessened.

To limit or lessen renal congestion, leeching and cupping are often employed. Although unnecessary in the milder cases, they are especially indicated in those in which there is the most congestion; those in which the urine is scant, very bloody; and in which there is backache.

Cathartics are also employed to deplete the renal vessels. Aperients, such as Rochelle salts and magnesia sulphate, are the best. In acute nephritis, and especially at first, catharsis must not be carried so far as to increase a patient's weakness. It is usually sufficient to maintain the stools soft, and defecation should take place only two or three times daily. A small glass of Hunyadi Janos water, a little of liquid citrate of magnesia or Carlsbad salts, taken in the morning, will usually accomplish this. If the urine is suddenly almost suppressed, a few liquid stools will often relieve the congestion which causes it, and should be provoked unhesitatingly.

It goes without saying that drugs and beverages, which in themselves are renal irritants, must not be used. Among these are all alcoholic beverages, tea and coffee, terebinthins, copaiba, and squills.

The obstructions in the renal tubules are composed of cells, the granular detritus which results from their dissolution, and hyaline substance. Much of this can be washed from the tubules by a more copious flow of urine. The hyaline substance has been shown by Purdy to be soluble in alkaline solutions; therefore, to make the tubules patent, the urine must be kept alkaline and must be abundant.

Digitalis will increase the general blood-pressure, and therefore the pressure in the glomerular tufts, by increasing the vigor of the heart's action and by contracting the arterioles. This will cause a more rapid filtration of water from the blood into the urinary channels in cases of acute nephritis. The quantity of urine is decidedly increased by this drug. Its action is preferable to that of strophanthus, for the latter, in therapeutic doses, does not contract the arterioles. The action of digitalis upon the arterioles is, moreover, valuable, since it lessens hæmorrhage. In order to render the urine alkaline, such drugs as the citrate and acetate of potash and liquor ammonii acetatis may be used. The first are the most agreeable to take, and may be employed in doses of from 2 to 4 grammes ($\frac{1}{2}$ to 1 drachm), repeated every two to four hours, as may be needed to keep the urine alkaline. Liquor ammonii acetatis may be given in doses of from 2 to 4 cubic centimetres ($\frac{1}{2}$ to 1 drachm). It is certainly true that, under the influence of these drugs, casts usually become less numerous and less perfect in outline.

The diuretics just mentioned are useful not only to keep the urinary tubules open, but also to promote a more perfect excretion of urinary solids. Digitalis helps by making a more copious flow of urine; the potassium salts aid by increasing the oxidation within the system and by stimulating the renal epithelium to greater functional activity. Therefore, larger amounts of urea are voided, which means that many imperfectly oxidized products of metabolism become perfectly oxidized, and are in a condition for easy elimination. This will greatly lessen the danger of uræmia. Although lactose and glucose are often very efficacious diuretics in cardiac affections, they are not so in renal diseases. Indeed, in the latter diseases they sometimes produce no appreciable diuresis. They probably act upon the renal epithelium, and, if it is not intact, they are inefficacious. In my own experience they have proved moderately efficient in some mild cases of chronic nephritis, but almost useless in the acute disease. Unfortunately, the epithelium is often so extensively desquamated or diseased that these drugs are able to promote only a very moderate increase of urine, urea, etc. We must depend, therefore, upon other channels to eliminate the poisonous effete materials that may be accumulating in the system. The skin will eliminate the largest amount of such matter; therefore, diaphoresis should be provoked early in all cases in which the amount of urinary solids voided daily is much reduced. (See page 269.) It should be repeated for a few minutes daily, or every second or third day, according as the kidneys are able to do their work more or less well. Diaphoresis must be resorted to with caution whenever there is much œdema, for, by provoking a sudden re-absorption of œdematous fluids which contain uræmic poisons, an attack of uræmia may be precipitated. It is safer first to remove the œdema by tapping or puncture. Drastics are important aids in preventing uræmia. They must be used with the cautions described on page 270.

General œdema, if it is not great, can be made to disappear by diuretics, drastics, diaphoretics, or all combined. If it is great, the last group of drugs are the most efficacious, but, as has just been explained, must be used with caution. Sometimes ascites and pleural and pericardial effusions can be removed by the same means, but usually less promptly and less perfectly. The serous sacs, often, must be drained. Rapid drainage of the smaller ones is best accomplished by an aspirator, and of the larger by a trocar. If diuretics, and occasionally drastics, or, instead, a hot-air bath is given, refilling of the sacs can be prevented. If general œdema is great, and if uræmic symptoms are present or feared, it is best to withdraw the dropsical interstitial fluid through deep ankle incisions. These are made preferably over the inner malleoli. They should be deep enough to incise all the tissues down to the periosteum, and should be at least three-fourths of an inch long. After the incisions are made the patient should sit erect, or be placed in a semi-reclining posture, with the feet lowered as much as possible, that the fluids may gravitate to them and flow freely from the wounds. In twenty-four hours all the interstitial dropsy can usually be removed from the body, and, by other means, it can be prevented from re-accumulating.

During the period of recovery the greatest care must be taken to prevent exacerbations by a regulated diet, by rest, and by careful clothing, so that the skin's temperature will be kept equable. During convalescence it is often advisable to send patients from our raw and changeable winter and spring climate to a warmer and more equable one, such as can be found in Florida, Georgia, and Southwestern Texas.

Iron, strychnia, and other bitter tonics are now most useful. The iron will cure the anæmia, which is incidental to the disease, and it helps to prevent and counteract the degeneration of renal epithelium. Strychnia is perhaps the most powerful stimulant of nutrition that we possess. Quinine and, to a less extent, other bitters, like gentian, act in the same way.

So soon as the kidneys perform their function fairly diuretics can be gradually omitted. Digitalis can usually be discontinued before the alkaline diuretics are, as they may be longer needed to maintain the urine alkaline, the tubules free, and the elimination of nitrogenous matter abundant.

The urine should be examined daily during the acute period of the disease, and particularly with reference to the amount of nitrogenous matter that is eliminated. During convalescence it need not be examined so often, but should be occasionally for some weeks, even after albumen has disappeared from it. So long as there is albuminuria exercise should be forbidden, although, in a good climate and favorable seasons, carriage-riding may be permitted during convalescence, and the patient may be moved from room to room, so that the mind may be kept buoyant by variety and change.

Prognosis.—The prognosis of acute nephritis is generally favorable, for almost all cases recover. It is usually considered less severe in later life than in early manhood or childhood. The danger to life is from uræmia and various complications. If the urine is very scant the case must be regarded as grave, because of the danger of uræmia. If pneumonia or empyema develop, death is almost certain. Extensive dropsy increases the gravity of a case, especially if the pleural or pericardial sacs are involved. A small proportion of cases become chronic. If the acute nephritis is secondary, the character and gravity of the primary disease must modify or shape a prognosis.

CHAPTER XXXII.

CHRONIC PARENCHYMATOUS NEPHRITIS.

Causes.—Chronic parenchymatous nephritis often follows acute, but oftener it begins as a chronic disease. Scarlatina is a frequent cause of it. Chronic suppuration may produce chronic nephritis alone or combined with amyloid infiltration. Malaria and syphilis are two diseases out of which it often develops. The uric-acid diathesis, gout, and rheumatism are also causes. The constant use of alcohol, and especially of the stronger beverages, predisposes to the disease, and sometimes undoubtedly provokes it. Many cases arise without assignable cause. Exposure to wet and cold and changeable atmospheres is regarded by some as provocative of many of these cases. I must agree with Ralfe in believing that work upon damp and cold ground, or a residence in rooms that are damp and cold, is much oftener a cause.

The disease occurs most frequently in males and during the first half of adult life. It is rare in childhood and of occasional occurrence in advanced life.

Anatomy.—When the disease attains its full maturity a lesion is developed that differs greatly from that of its early period. These differing states may be described as its first and second stages.

In the first stage the kidneys are much enlarged. They may be two or three times larger than natural. The capsule is smooth, and can be readily stripped from the kidney's substance. When the organ is much enlarged it will gape through an incision in the capsule. The surface of the organ is usually mottled gray, or often almost white and red. These colors may exist in varying proportion. Many times the red areas are pale red. In numerous cases the kidney's surface is uniformly white or yellow-white. When a section is made through the kidney the enlargement will be seen to be due to a broadening of the cortex. It is often twice its usual size. The cut surface of the cortex will be mottled or uniformly yellowish white, as is the surface of the organ. The pyramids are red or, rarely, pale. Usually, there is a strong contrast of color between the cortex and pyramids. The yellow color of the kidney is due to fatty degeneration. This is the most characteristic renal change in this disease. The microscope will reveal the degeneration most clearly. The epithelium of the renal tubules is the focus of the degeneration. Its cells are often swollen, very granular, and contain numerous visible droplets of fat. When swollen they may nearly occlude a tubule. They frequently disintegrate, and fill the tubules with a granular detritus and oil-droplets. When the cells are cast off they are usually replaced by new ones that are thin, and, therefore, enlarge the calibre of the tubules. These changes are most marked in the convoluted tubules, but are observable, also, in the others. Within the tubules hyaline casts are abundant, and granular matter, desquamated epithelium, occasionally leucocytes, and red blood-corpuscles exist in varying amounts. The glomeruli are usually not changed in size, but sometimes they are a little enlarged. Many of them are normal in appearance; many of the others have thickened capsules. The capsular epithelium proliferates and causes the thickening. Sometimes this thickened tissue becomes partly or wholly homogeneous or hyaline. Fatty degeneration is most common in these cells. The epithelium covering the capillary tuft is also thickened. Hæmorrhages sometimes take place into the glomeruli, and also into the intertubular tissue. In the rare cases, which are called chronic hæmorrhagic nephritis, they are abundant and almost constant. The tuft of capillaries sometimes contracts or atrophies, and may, in part or wholly, become hyaline. The stroma of the kidney always contains a serous exudate, but in places, especially near the glomeruli and about interlobular veins, there are cellular exudates. Sometimes, in the cells of the interstitial tissue, droplets of fat can be seen.

In the second stage the kidney is not so large or, in the most marked cases, contracted. Its surface is granular or rough, at least in spots, and sometimes generally. It is mottled or uniformly yellowish white. The capsule is adherent where the surface is contracted. The cut surface of the cortex exhibits an irregular outline, and shows that it is a cortical contraction that causes the general renal contraction. Under the microscope the points of greatest contraction will be seen to be composed chiefly of connective tissue. The tubules are contracted, atrophied, and often obliterated. The glomeruli are small; their capillary tufts are much contracted. The glomerular capsule is thick. Both tuft and capsule are often homogeneous and hyaline. Between these areas of contraction are others that exhibit the lesions of the earlier stage. The contracted areas are usually near the surface of the kidney and in the interlobular districts. A desquamation of epithelium from the tubules, which then collapse and are obliterated, precedes the proliferation of connective tissue and disappearance of normal renal structure. The interstitial tissues now proliferate, and the exuded leucocytes help to produce new tissue, in the process of whose development adhesions form with the renal capsules. The interstitial changes lead to compression of neighboring tubules and to interference with the capillary circulation. This in turn leads to more destruction of epithelium and an extension of the lesion of induration. Such changes occur only in the most chronic cases.

Besides these characteristic renal lesions, œdema of the subcutaneous tissue and of the serous sacs is common, and less frequently œdema of the lungs or glottis occurs. Cardiac hypertrophy is common. It may be either unilateral or bilateral. Valvular lesions of the heart occur in about 25 per cent. of cases.

It is quite evident, from the nature of these pathological changes, that diuresis must be lessened in the first stage, but in the second it may be increased, because of the interference with the circulation, and consequent increased arterial pressure which the cirrhosis produces. On account of the destruction of epithelium or interference with its function, there is a lessening of excretion of urinary solids in both stages. Thus, the same functional derangements that exist in acute nephritis are produced, but, because of the insidious and slow development of chronic nephritis, its clinical, as well as its pathological, picture differs greatly from that of acute nephritis.

Symptoms.—Chronic nephritis may develop from acute, and is especially apt to when scarlet fever is the primary cause of the nephritis. But its most characteristic type begins as a chronic affection. When it develops from the acute, red corpuscles gradually disappear from the urine, but the albumen in it may increase. In varying amounts there are, also, casts, cells and their detritus, and oil-droplets either floating in the urine or imbedded in the casts and cells. Œdema persists, or, at times, disappears partly or wholly, to re-appear occasionally.

When its onset is insidious the disease may be discovered by a chance urinalysis. The patient may complain only of weakness, of anorexia, of asthma, or some symptom of chronic uræmia. Quite as often attention is attracted to the condition of the kidneys by the development of œdema. The latter is oftenest first noticed in puffed eyelids in the morning, or swollen ankles at night. When the disease is chronic from its inception, dropsy may not develop until it has been established for weeks or months. At first, it may fluctuate, disappear for a few days, to re-appear, or lessen, and again increase. When once developed, it rarely disappears entirely or for long. As a rule, in spite of temporary abatement by treatment, it will increase and distend the subcutaneous tissue over the whole body. The abdominal cavity is generally filled, and sometimes the pleural or pericardial cavities are also. When œdema is great the scrotum is usually distended and the skin of the penis much swollen and deformed. The legs will become so large that the tightly-stretched skin is glass-like in smoothness. Great stretching of the skin usually causes malnutrition, and eczema breaks out upon it. Occasionally, gangrene will occur, sloughs will form, and leave deep and very sluggish ulcers. Not unfrequently from an eczematous surface upon the legs, and oftener from deep cutaneous ulcers, the serum will flow so rapidly as to drain the tissues of their fluid and cause a partial or complete disappearance of dropsy. Eczema may attack other parts of the body, but the legs and scrotum are its favorite sites. Ascites is often so great that it compresses the abdominal viscera and prevents hearty eating even when there is an appetite. It crowds the diaphragm upward, often displaces the heart and prevents a free expansion of the lungs. Ascitic patients are, therefore, short-winded. They frequently avoid the reclining posture, because the fluid gravitates against the diaphragm and makes respiration painfully short and labored. When the second stage of the disease develops, the increased diuresis sometimes causes the dropsy to disappear. In rare cases of chronic parenchymatous nephritis, dropsy does not develop; but it occurs more uniformly and more persistently in it than in any other form of renal disease. Its amount varies inversely as the quantity of urine varies.

As in other forms of nephritis, the urine furnishes the most pathognomonic signs of the malady. In the first stage of this disease the urine is diminished in amount. This diminution may be moderate, or it may be to from 150 to 350 cubic centimetres (6 to 12 ounces) a day. It is usually dark colored and turbid. Its specific gravity varies from 1020 to 1040 and is usually greater than is normal. Its reaction is acid. Albumen is abundant in it. Commonly, it amounts to about 1 per cent., but may be more than 5 per cent. If the disease develop insidiously, the amount of albumen is at first small and gradually increases. There is an abundant sediment in the urine. This contains urates; often a few red corpuscles from the blood; granular and fatty epithelial cells, which are always present, and are at times very abundant; casts and some granular matter, intermingled with which oil-drops can generally be discovered. The casts at first are long, narrow, hyaline, or granular; later, they are shorter, broader, and more granular and fatty. Sometimes a few granular or fatty epithelial cells will adhere to them. The presence of oil-droplets, or fatty cells or casts, is particularly characteristic of this form of nephritis. Red corpuscles from the blood are often absent in individual specimens, but may be very abundant when so-called acute exacerbations occur. Then the urine diminishes still more in amount, becomes red and cloudy, of high specific gravity, and abundantly albuminous. It resembles very closely the urine of acute nephritis, but contains more oil and fatty matter.

The urinary solids are always diminished in amount. Especially is urea diminished; chlorides are less so, and phosphates and sulphates least. The percentage of urea in single samples of urine is often increased, although the total eliminated in twenty-four hours is much diminished. The amount of urea voided daily will vary. It may be normal for some days, and then may be diminished.

In the second stage of the disease, the quantity of urine voided daily is commonly normal, or more than normal. It is lighter colored and clearer than in the earlier stage. Its specific gravity will vary from 1010 to 1015. The quantity of albumen lessens, and the daily excretion of urea is still more diminished. These urinary changes are characteristic of contracted kidney. The formed elements in the urine are less abundant than in the early stage, but like them.

In the first stage the pulse is usually small and soft. It may be quicker than normal if the patient is feeble, excited, or hurried. The heart is usually, at first, not hypertrophied or dilated. Later, one or both changes will occur in the left ventricle, and sometimes in both ventricles. If the kidney become hardened and contracted and the blood's circulation through it impeded, an hypertrophy of the left ventricle occurs, and usually moderate dilatation. But even when the kidney does not contract, if the patient grow feeble and œdema is considerable, some, and at times very great, dilatation of the ventricles takes place without hypertrophy. Sometimes hypertrophy of the left ventricle occurs in this disease without assignable cause, unless there are irritants in the blood that may provoke it. Hypertrophy of the right ventricle is probably due to failure on the part of the left to maintain an equilibrium between the arterial and venous circulation. This causes passive engorgement of the lungs, which must be overcome by the right ventricle. When the heart is dilated and feeble clots are liable to form in it, and may cause the various phenomena of cardiac thrombosis. The state of the blood in this disease predisposes to thrombosis. It contains a larger proportion of water and fibrin-makers than is normal; a diminished number of corpuscles and albumen; a somewhat increased amount of fats and salts. The quantity of urea in it varies inversely with the power of the kidneys and skin to eliminate it. The anæmic condition of the blood sometimes produces cardiac murmurs. Accidental murmurs rarely occur as the result of cardiac dilatation independently of the anæmia, and still more rarely they are due to interference with the action of the valves by thrombi formed on them or their muscular papillæ.

In the second stage of the disease the pulse becomes hard. The physical signs of cardiac hypertrophy are demonstrable. If an enlargement of the heart cannot be shown to exist, an unusually forceful apex-beat, and often one displaced downward and to the left, and an accentuation of the second sound over the aorta are proof of it. Hæmorrhages may occur from the nose and other mucous membranes. The state of the blood and a cardiac hypertrophy will dispose to them. Albuminuric retinitis (see page 321), while possible, is not common, and is especially rare in the first stage of the disease. The temperature is normal, unless some complicating inflammation causes it to rise.

The skin is usually dry and rough. Though it is pale, it has not the clear, white color that is often seen in acute nephritis. It is yellowish or parchment-like. This is especially true of the most chronic cases.

A gradual but marked loss of strength and flesh takes place from the first. During periods of remission in the course of the disease some gain may occur, but such gains are only temporary. Emaciation is often masked by dropsy. It is very evident when the latter is removed. Loss of strength will, in time, confine a patient to the house and to the bed. Respiration will not be interfered with, except by œdemas, such as excessive ascites; pleural, pulmonary, or laryngeal dropsy. Chronic uræmia may cause asthma.

Lack of appetite is an early and usually persistent symptom. Nausea and vomiting are sometimes due to indigestion, but often to uræmia. When œdema is great, a serous fluid is sometimes vomited, which is dropsical in origin. In such cases there is often a serous diarrhœa, which is also due to intestinal œdema. Rarely, intestinal ulceration complicates chronic nephritis. In many cases, although there is little appetite for food, there is no demonstrable failure to digest what is taken. If vomiting and nausea occur, unassociated with evidences of indigestion, they are usually due to uræmia. Recently, Biernacki has studied gastric digestion with care, in cases of Bright's disease. He finds free hydrochloric acid in the stomach in diminished amount, and sometimes wholly absent; pepsin apparently diminished, and lactic acid in only small quantities. These changes occurred both with and without symptoms of indigestion.

A lack of energy and ambition is as evident as a lack of muscular strength, even in the beginnings of the disease. Acute uræmia occurs less frequently in chronic parenchymatous nephritis than in other renal inflammations. Mild chronic uræmia is not very uncommon. If severe uræmia occur, it is usually when there are acute exacerbations of the nephritis, or toward the close of life. In the second stage uræmia is relatively frequent, but it is not so common as in interstitial nephritis. It is probable that several factors contribute to produce amenity to uræmia. The diminished appetite and disinclination for albuminous foods help to prevent the formation of uræmic poisons. The slow waste of the tissues and disinclination for active exertion prevent the rapid formation of them from the living tissues. Much urea and presumably other effete and toxic matter is stored in the dropsical accumulations which are usually so abundant.

The second stage of the disease is characterized by an increased flow of watery urine, by a disappearance of dropsy, by a pulse of high tension, cardiac hypertrophy, and by relatively frequent uræmia.

In the course of chronic parenchymatous nephritis, exacerbations often occur which closely resemble attacks of acute renal inflammation. They occur oftenest in those cases that were at first acute, or in those that are sometimes denominated chronic hæmorrhagic nephritis. The latter are characterized pathologically by a mottling of the surface and interior of the cortex with red and yellow areas, and by numerous minute points of hæmorrhage. Undoubtedly, exposure to cold and dampness often precipitates these attacks.

The disease may last for months or even years. Recovery is very rare. The longer it lasts, the less are the chances of recovery. Few cases exceed two years in their duration, unless renal contraction occurs. They may then be more protracted. A larger proportion do not exceed one year. Death may result from uræmia suddenly, from dropsy which disables one of the organs essential to life, or from complicating inflammations like pleurisy or pneumonia.

Diagnosis.—A direct diagnosis is usually possible. It is based (1) upon the character of the urine, (2) upon the character of the anæmia, (3) upon more or less constant and usually considerable œdema, and (4) upon the chronicity of the disease. It must, sometimes, be differentiated from acute renal inflammation, from amyloid kidney, and the second stage from interstitial nephritis. It is chiefly apt to be confounded with acute nephritis when so-called acute exacerbations occur, or when acute cases tend to become chronic. Acute exacerbations are distinguished from acute nephritis by the history of prior œdema, or of other symptoms of nephritis, and by the existence in the urine of fatty cells and casts, and often free oil-droplets. Usually, the complexion is different. It is a purer white in the acute disease. If the kidney is amyloid only, the urine may be undiminished in amount, or even increased, and will contain abundant albumen. The most significant symptoms of lardaceous kidney are, besides the existence of a cause for the lesion, the co-existence of enlargement and hardening of the spleen and liver. When, as not unfrequently happens, chronic nephritis and amyloid infiltration co-exist, a diagnosis of both lesions may be impossible. It is only necessary to distinguish chronic parenchymatous nephritis from interstitial when the former has reached its second stage. The urine may be very similar from both these lesions, though usually from the former it is less abundant, more albuminous, and of higher specific gravity. In the cases of parenchymatous nephritis there is a history of former œdema, which is rare in interstitial nephritis, except in the last stage. The diagnosis will, therefore, depend chiefly upon the history of the development of the disease.

Treatment.—Whenever it is possible, the cause of chronic nephritis should be removed. This can be done by draining abscesses, by treating syphilis or malaria, by discontinuing the use of alcohol, or other renal irritants.

If the disease begin as acute nephritis, it must be treated as the latter should be: To maintain the permeability of the uriniferous tubules the urine should be kept alkaline; to prevent renal congestion and cardiac hypertrophy, or to lessen œdema, the circulation should be equalized by laxatives; to lessen œdema, and especially to prevent uræmia, hot-air baths and rest should be relied upon; to maintain nutrition, and to prompt the kidneys to greater activity, a milk diet should be maintained; eggs and meat should be forbidden. Such a course of treatment will usually lead to marked improvement, and often even to recovery. During convalescence, patients must be carefully guarded against relapse. Its possibility must be remembered even for a year or more, and it should be averted by prophylactic measures similar to those that are necessary in the course of the disease to avoid acute exacerbations.

If the disease is chronic from the first, the indications are: (1) to guard against acute exacerbations, (2) to prevent or limit fatty degeneration, (3) to lessen the excretion of albumen when it is excessive, (4) to prevent uræmia, and (5) to lessen œdema. If the second stage is reached, we may attempt to lessen (1) the interstitial hyperplasia which characterizes it, and (2) cardiac hypertrophy.

Acute exacerbations can be best avoided by clothing those who have chronic nephritis in woolen or other underwear that will maintain an equable surface temperature for the body, or at least prevent sudden surface changes. A residence in an equable climate is extremely desirable. Patients should especially avoid damp and cold climates, and houses that are damp or upon soil imperfectly drained. All substances that, by elimination through the kidneys, irritate them should be avoided. A milk diet is, in most cases, almost a specific, so favorably does it influence the disease.

The most characteristic feature of the lesion is fatty degeneration. A milk diet, or, at least, one easily digested and assimilated, is essential to prevent this, by maintaining a healthful nutrition of the renal cells. Thorough oxygenation of the blood is just as necessary for the maintenance of perfect tissue-change. Therefore, the rooms of the sick should be perfectly ventilated, and, if respiration is interfered with, or uræmia evident or threatening, oxygen inhalations may possibly help both to cleanse the blood and make metabolism more perfect. Of drugs, the most important is iron. It seems particularly to limit or prevent degeneration. Its preparations are so numerous that there is a wide field for choice. The preparations which I oftenest use are the citrate, the potassio-tartrate, the subcarbonate, the iodide, and the tincture of the chloride. Occasionally a change should be made from one to another of these preparations. If the anæmia does not promptly disappear, or the evidences of disease lessen, courage should not be lost so long as the symptoms do not become more grave, for changes are slowly wrought, in so chronic a trouble, and the remedies must be perseveringly used. Iron acts best when given with strychnia, or quinia, or both. They undoubtedly stimulate cells to greater nutritive activity. A capsule can be given, three or four times daily, that will contain these tonics and iron in combination, or a solution of them may be administered. They should be given in varied forms, but almost constantly, for months.

In this variety of nephritis the loss of albumen is considerable, and may contribute materially to cause weakness. Various drugs are used to lessen it. Some of these, I am convinced, accomplish the object, but I have not seen sufficient improvement produced in the general health of a patient, while they are used and the albuminuria is checked, to make me confident of their utility. The drugs which can be most safely and efficiently employed to limit the excretion of albumen are: Tannate of sodium, which can be given in doses of from $\frac{1}{3}$ to $1\frac{1}{2}$ grammes (5 to 20 grains), in water; tannic or gallic acid, in their usual doses; or nitric acid, ergot, caffeine, or fuchsin. The last is given, in doses of from $\frac{1}{2}$ to 2 decigrammes (1 to 3 grains), in pills.

I have already described fully the methods by which uræmia is to be avoided and treated. I need now only outline the treatment most applicable to this form of nephritis. If œdema is considerable, a patient should remain in bed, or only be moved to a lounge or another bed. If there is no, or very little, œdema, moderate exercise may be permitted; but it should never be exhausting or very long continued, for much exercise certainly increases the danger of uræmia.

A prescribed diet is most important in the treatment of this affection, for by a careful regulation of it the danger of uræmia can be greatly lessened. Milk is, for chronic parenchymatous nephritis, more than a food; it is curative. Just how it produces its good effects is not known. It is readily digested and converted into very assimilable nutriment. It contains very little of waste or useless matter. It is also diuretic. These are qualities which adapt it especially to the disease. It contains a small amount of albumen, but not enough to be dangerous, unless the daily volume of urine is very greatly reduced. Milk, as an exclusive food, contains an excess of fats. This is undoubtedly one reason why it is so perfect a food, although it contains so little albumen, for it is proven that less albumen is needed in proportion as fats and carbohydrates are taken in increased amounts. A strictly milk diet, long continued, has often proved curative. Disgust for milk is frequently caused by restricting patients too quickly to a diet of it only. Therefore, the regimen should be restricted somewhat gradually. Meats, eggs, fish, cheese, and leguminous vegetables should be first omitted, and slowly the farinaceous foods can be withdrawn. A milk diet is best tolerated when the beverage is taken in moderate amounts every two or three hours. It is also important that the stomach should not be overloaded, as indigestion, which is likely to result from it, usually produces substances that irritate the kidneys in the process of their elimination, or help to make the blood toxic. If a patient can, he should adhere to a milk diet. Often, however, patients grow wearied of it. They can occasionally be allowed a little fruit, or a soup made of milk, flavored with some vegetable, like asparagus, pease, or tomatoes. Occasionally, thin milk-gruels, apple-sauce and milk, or some other fruit and milk, can be similarly employed to vary the diet. Upon a strictly milk diet, a patient should partake of it at about seven and ten in the morning; one, four, seven, and ten in the afternoon and evening. If it is varied by some of the articles just mentioned, they may be substituted at ten and four, or at seven in the morning and seven in the evening, while milk only should be taken at other times. But it is best to adhere as long as possible, and in as many cases as possible, to a purely milk diet. Often, if the regimen is varied for a few days, as I have suggested, a milk diet can be again adopted by the patient without distaste. When milk is altogether distasteful, and cannot be taken in quantities sufficient to maintain nutrition, a farinaceous and fruit diet must be adopted, but all albuminous food should be excluded from it. As pease and beans contain considerable amounts of nitrogenous matter, they should not be used. If milk is not taken freely, water should be. It helps to produce freer diuresis. It dilutes toxic matter that may be in the blood. Distilled water or spring water that is as free of mineral matter as possible is the best, as it is able to dissolve more waste products, especially those imperfectly prepared for solution, and therefore most apt to accumulate in the system. The use of large amounts of water at springs, and the good results which accrue, have made many famous for the treatment of renal diseases. At least, a glassful of water should be taken at each meal-time, and one between meals and at bed-time. If patients cannot be kept on a milk diet, but must be allowed one that is more varied, still greater care must be exercised that the stomach be not overcrowded, or indigestion produced. Only so much as can be well digested and assimilated should be taken, and it should be washed into the blood with frequent drafts of water.

Diuretics may be relied upon to prevent uræmia if the daily quantity of urine voided and of nitrogenous matter eliminated is lessened a little only. In more urgent cases they may be aided by occasional purges or by sweatings. Diaphoresis, it must be remembered, should be provoked with care, or not at all, if œdema is great.

To relieve dropsy reliance must again be placed upon diuretics, cathartics, and diaphoretics. If pleural, pericardial, or peritoneal dropsy endanger life by compressing the lungs or the heart, it should be relieved by aspiration, or drainage through a trocar. If subcutaneous œdema is great, and it does not seem safe to remove it by diaphoresis, incisions at the ankle may be made, and the whole may be withdrawn. When œdema is great the amount of urine voided daily is very small, but often when the œdematous fluid is rapidly withdrawn through incisions it will increase almost to a normal quantity. The kidneys renew their activity, and by careful treatment free diuresis can often be maintained for a long time. Perfect drainage of the subcutaneous tissues is usually accomplished by ankle incisions in twenty-four to forty-eight hours. The incisions generally heal in three or four days after the drainage ceases. The procedure may be repeated several times without ill effects. The drainage of the tissues is less likely to be complete if the anasarca has been of long standing, or if the dropsy has often re-appeared. The connective tissues in which the fluid accumulates gradually proliferate, and the intercommunication of lacunar spaces and lymphatic channels becomes less perfect.

If the second stage of the disease is developing, mercurials and the iodides may be used to check the connective-tissue proliferations. I doubt the efficacy of these drugs in cases that have not a syphilitic origin, and even in the latter they are not always successful. They are both diuretic, and may aid patients by virtue of that property. The iodides lower arterial pressure by dilating the arterioles, and thus may delay cardiac hypertrophy, dilatation, and final exhaustion. The nitrites produce prompter and greater arterial relaxation, and, therefore, are often resorted to in this stage of the disease to relieve the heart of overwork. (See pages 218, 228.) When the kidney contracts there is greater danger of uræmia than in the earlier stage. It must be averted by the same means as in other cases. The danger most peculiar to this stage of the disease is cardiac exhaustion from overwork, and this must be prevented, if possible.

Complications, such as pleurisy, pneumonia, and endocarditis, must be treated as they would be under other circumstances. While many cases of chronic parenchymatous nephritis are incurable, life can often be prolonged for even many years by careful treatment, which will prevent fatal complications.

CHAPTER XXXIII.

INTERSTITIAL NEPHRITIS.

Causes.—Interstitial nephritis, although it may occur in infancy or childhood, is extremely rare, except in the last third of life. Deaths from it are most frequent between the fortieth and sixtieth years, and especially between the fiftieth and sixtieth years. It is, however, so eminently a chronic affection that its beginning must be dated back from five to ten years, or longer. One form of contracted kidney is associated with general arterio-sclerosis, and is one of the lesions common to old age.

Renal cirrhosis is three or four times commoner in men than in women. It undoubtedly occurs oftener among high livers than among those whose diet is simple. The harm is probably done by the extractives and spices which are so abundant in game, in richly-dressed meats and soups, such as constitute a large part of the regimen of good livers. These substances, not easily metamorphosed in the blood or tissues, are irritants to the kidneys, by which they must be eliminated. Moreover, those who habitually live upon such foods are usually dyspeptics, and, therefore, are only able to prepare them imperfectly for assimilation. In most instances, such eaters do not drink freely of water, which, if taken copiously, might wash these substances rapidly from the system, and not leave them long enough in it to cause prolonged irritation. The beverages oftenest used by them are the alcoholics, which are undoubted renal irritants, and most certainly dispose to fatty degeneration of the epithelium. Writers upon this subject almost unanimously declare that the excessive use of alcoholics is the commonest cause of cirrhotic kidney. Renal cirrhosis usually occurs in those who use these beverages steadily, but moderately. The excessive use of alcoholic drinks leads often to acute or oftenest to chronic parenchymatous nephritis. The pathological statistics collated by Formad prove this. My own clinical observations confirm it.

Gout and the uric-acid diathesis are often primary to renal cirrhosis. The slow elimination by the kidneys of irritating nitrogenous substances imperfectly prepared for conversion into urea and eliminable matter is the probable cause of the nephritis.

Chronic plumbism occasionally produces renal cirrhosis. Scarlet fever and malaria are rarely primary to this form of nephritis. It is sometimes said that prolonged mental depression disposes to this renal lesion. It is true that the mental and renal trouble often co-exist. But it is not demonstrated that there is a relation of cause and effect between them. Depressed mental states are common to dyspeptics, and also to those having uric-acid diathesis. It is more probable that the mental and renal state have a common cause than that one is the cause of the other.

In many, though not in a large number of cases, heredity is seemingly an etiological factor. Renal cirrhosis is commonest in temperate climates. It rarely grows out of acute nephritis. If the latter almost subside, but persist as a small islet of chronic inflammation, interstitial nephritis may be the result. It also grows out of chronic pyelitis, and through it, indirectly, out of chronic cystitis and urethritis.

Anatomy.—The renal cirrhosis which results from arterio-sclerosis is a lesion quite distinct from that which results from gout, plumbism, high living, and other causes. When a renal arteriole is sclerosed and finally becomes nearly or quite impermeable, the glomerule or group of glomeruli to which it furnishes afferent vessels contracts. At first the capillary loops become shrunken, hyaline, and impermeable. The glomerular epithelium is shed, and partly or wholly disappears. The capsule contracts around the small homogeneous mass which represents the former capillary tuft. The capsule sometimes is thickened, but often remains unchanged. Such glomeruli are frequently one-fourth to one-sixth their normal size, and are functionally useless. When a glomerulus ceases to pour fluid down its uriniferous tubule the latter also contracts. It at first collapses, its epithelial cells then diminish in size. They often fill the contracted tubule. Sometimes a tubule will be converted into a cyst by an obstructing plug of colloidal matter. There may be no interstitial change, but usually small, abnormal islets of connective tissue are discernible about the sclerosed arteries. These minute changes cause an irregular contraction of the renal cortex and make the surface rough or granular. The kidney does not become as tough and hard as in cases of interstitial hyperplasia.

When there is true interstitial nephritis, the kidneys are imbedded in large amounts of fat. They are contracted, but not equally. The surface is rough and granular, reddish brown, or rarely grayish brown. Cysts, from the size of a pin-head to a bean, are common, both on the surface and in the renal substance. They are distended with a clear fluid. The kidney is firm and leathery in consistence. The capsule is thick and firmly adherent to the renal substance at the points of depression on its surface. A section of the organ exhibits the same color as its surface. The granulations on the surface and the tissue beneath them are dark colored. The depressed areas may be grayish. The cortex has a very irregular width, and is often in places very narrow. It may be to the pyramids as one to five or as one to six. The pyramids are usually deeply congested. The pelvis is often dilated, and sometimes inflamed. In the gouty kidney, gray, hard streaks can be seen and felt. These consist of deposits of urates in the interstitial tissue, the epithelium, or within the tubules.

Under the microscope the thickening of the capsule is seen to be due to a connective-tissue hyperplasia, which is greatest at the points of depression on the kidney's surface. At these points the cirrhotic renal tissue and new capsular tissue are confluent, and, therefore, united. The areas of cirrhosis are irregularly disposed in the cortical substance and, usually united to a greater or less extent, encircle more normal territories. In the centre of the cirrhotic tissue renal structures cannot be seen. Fibrous tissue only composes it. Toward the periphery of these areas their mode of extension can be studied. The glomeruli are seen to be greatly contracted or in process of contraction. They may be not more than one-eighth of their normal dimensions. Their capsules are usually enormously thickened by concentric layers of fibrous tissue. The capillaries may have atrophied and disappeared, or may be represented by a small homogeneous, granular, or hyaline mass. No glomerular epithelium can be seen. Occasionally, a greatly dilated and cystic glomerulus may be observed. The tubules are seen to be contracted, the epithelium lining them to be atrophying, or sometimes wholly disintegrated. In the latter case the tubule is outlined only by its basement membrane. The tubules then become obliterated. As they contract and disappear the tissue between them increases in amount. It is fibrous and abundant. Here and there a few round cells and embryonic connective tissue cells can be seen. The tubules that are least affected are often unusually tortuous. They look nearly normal in places, and elsewhere exhibit the changes which are seen in chronic parenchymatous nephritis. Such changes develop very gradually. The atrophy of the tubules is partly due to disturbance of nutrition, caused by the interstitial changes, and partly by the destruction of glomeruli. When tubules become obstructed in part of their course only, they may expand and form cysts. Many arterioles, especially in the cirrhotic areas, are made useless by endarteritis obliterans. Usually, the entire arterial wall is thickened, but its intima is especially so, and the calibre of the arteriole is, therefore, almost or quite obliterated. In other cases, the tunica adventitia and muscular coat are thickened by the formation in them of masses of a waxy appearance. These changes also produce more or less vascular stenosis. Hæmorrhages into the kidney are not common. It is still a question whether the interstitial hyperplasia or the tubular atrophy is the primary lesion; whether the interstitial changes cause the tubules to contract and disappear, or whether, because of the disappearance of the latter, the former undergoes a compensatory hypertrophy.

Endarteritis obliterans and arterial sclerosis also often occur in other organs. The left ventricle of the heart is hypertrophied and generally dilated. Sometimes both ventricles are. Occasionally, points of fatty degeneration, or indurative degeneration, can be seen in the heart-muscle. In some cases, the endocardium is thickened in spots, or there may be evidence of endarteritis. There may also be thickening or evidence of inflammation of the pericardium, pleura, or peritoneum. The dura mater and arachnoid may also be thickened. Cerebral hæmorrhages are common complications of the disease. Pulmonary consumption occurs less frequently, but often. Gastric and intestinal catarrh are usual concomitants of the renal trouble.

Symptoms.—Clinically, three stages of the disease are recognizable; the first may be described as the state in which there is high arterial tension without recognizable cardiac hypertrophy; the second, one in which there is cardiac hypertrophy; and third, one in which there is cardiac failure. Death may occur, in either of these states, from intercurrent disease, uræmia, or cerebral hæmorrhage. Life is often prolonged for many years after the disease is established. The malady may last for twenty or more years, and commonly does for from five to ten.

It is extremely important that this disease should be recognized early, as the danger to life is much greater after the heart has hypertrophied; and by careful treatment in its early stage the advance to the later ones can be delayed. Unfortunately, the first stage is not always readily recognizable.

The onset of renal cirrhosis is always insidious. The early symptoms do not direct attention to the kidneys. In the first stage most of the symptoms are variable. They will exist in one case and not in another, or at different times in the same case. One of the most constant of these variable symptoms is indigestion. It is usually very persistent when it exists. It is characterized by flatulence, and rarely by pain and nausea. Another of these early symptoms is headache. It may affect any part of the head, but oftenest it is occipital and felt chiefly or only in the morning. In many cases there is hemicrania. These are undoubtedly often symptoms of mild uræmia. Vertigo is a common symptom. It is usually momentary, but is especially liable to recur. The character of the pulse is quite constant. It is fairly full and regular, but unusually tense. It feels cord-like under the finger, and is not readily compressible. In the sphygmogram the dicrotic notch is elevated from the respiratory line, and the line descending from the apex of the wave to the dicrotic notch is noticeably bowed upward. The urine is still more characteristic. Usually, it is made once or twice during the night, and in quantities abnormally large. This may be the first symptom that attracts the physician's attention to the condition of the kidneys. It does not alarm the patient, for frequent nocturnal micturition develops so slowly that he regards it as a habit, and not as a pathological condition. More urine is made in twenty-four hours than normal, but it is especially increased at night. It is usually limpid and clear, acid in reaction, and from 1014 to 1020 in specific gravity. It may be faintly albuminous at all times, or only after a hearty meal or violent exercise, and not in the early morning. But frequently it is altogether absent. On standing a precipitate may form, which is usually composed of crystals of calcium oxalate and uric acid, with sometimes a few hyaline casts. The quantity of urea voided in twenty-four hours is often less than normal.

With these symptoms we get the history of high living, or of some of the usual causes of the disease. If there is persistent high arterial tension and none of the other common causes of its existence, renal cirrhosis should be sought for. If high arterial tension co-exist, with the voiding of an unusually large amount of urine of low specific gravity, which contains a diminished proportion of urea, the existence of beginning renal cirrhosis may be looked upon as probable. If albumen is present in small amounts, constantly or occasionally, and if hyaline casts are found in the urine, the diagnosis may be made positively.

In the second stage the symptoms of the first are aggravated or intensified. Headache and vertigo are more common than in the first. If there is indigestion, it is more severe. Not only is there flatulence, but often nausea and vomiting. The gastritis is usually persistent if it exist, but varies greatly in intensity from time to time. The pulse is even more tense than in the earlier stage. The heart causes a strong apex-beat which can be seen and felt, but is somewhat lower and farther to the left than is normal. The cardiac sounds are normal. At the apex the first sound is strong. Over the aorta the second is accentuated. By percussion the area of dullness is demonstrably increased to the left. Occasionally, hæmorrhages from the nose, or into the retina, or within the brain, will occur. The urine is even more abundant, and more uniformly abundant, and made oftener at night than in the earlier stage. It is more constantly limpid and clear. Its specific gravity is lower,—from 1004 to 1014. It is, with rare exceptions, slightly albuminous. However, cases are observed in which albumen is never discoverable. Usually, it is, occasionally, if it is not constantly, present. It will be found most certainly in urine that is made in the evening, after a day's work, and after a meal. The daily elimination of urea is oftener less than in the first stage. As in the latter, the urine may contain no sediment, or, if any, a very small one. A few casts can occasionally be found in it. They are hyaline or granular, and may sometimes contain a few oil-droplets. Uric acid composes a part of the scant sediment. Cells are rarely found in it.

Respiration is usually normal. It is rarely dyspnœic from asthma which is caused by uræmia, or from pulmonary œdema. The bronchi and lungs are especially liable to various inflammations. In the first stage there may be no observable loss of strength, but in this endurance and, usually, strength are somewhat lessened. The skin is dry, rough, and parchment-like or gray, but not white or clearly anæmic, as in acute nephritis.

Nervous symptoms, usually of uræmic origin, are common. The dangerous ones are convulsions and coma. The less dangerous ones are disturbances of hearing, such as roaring, singing, or temporary deafness and insomnia, mental depression, various shifting neuralgias, and numbness. Uræmia is especially frequent in this stage of the disease. The eyesight is often impaired. It may be extensively affected by uræmic amaurosis, or what is called albuminuric retinitis. The first affection is usually temporary, although blindness may be complete. The blindness is generally sudden in onset. The pupils continue to react to light, and no changes are discoverable in the retina that are necessarily associated with the blindness. "Albuminuric retinitis" may be due (1) to œdema of the retina; (2) to degeneration in patches, which appear white, and are fatty or sclerotic; (3) to hæmorrhages; (4) to inflammation of the ocular end of the optic nerve; and (5) to atrophy of the retina and nerve, produced by some of these lesions. Often more than one of these lesions co-exist. They can only be diagnosed by an ophthalmoscopic examination. The subjective symptoms vary. Blindness may be gradually or suddenly developed as a result of them, or eyesight may be imperfect. All objects may be blurred, or only near ones or distant ones. Objects may appear distorted. Bright or dark spots may come before the eyes. Many of the symptoms are mitigated by time, especially those that arise suddenly; but they rarely disappear entirely.

In the third stage the prominent symptoms are those of cardiac exhaustion and failure. The hypertrophied heart is dilated, and is usually degenerated. The pulse grows soft, small, and quick. It often becomes irregular. The area of cardiac dullness is increased. The apex-beat is displaced to the left. It may be visible and somewhat forceful because of hypertrophy of the heart-muscle, although the latter is too weak to do its work. In other cases it can be faintly seen or felt. The cardiac sounds are not so strong as in the earlier stage, and especially does the first sound at the apex grow short and valve-like. But more significant than these cardiac changes are the diminishing excretion of urine and the developing œdema. The urine may be much reduced even below the normal amount. The total daily excretion of urea remains diminished. The specific gravity may increase, but almost uniformly remains below the normal. Albumen is usually a little more abundant. Casts oftener contain fat or oil. Œdema begins first about the legs, but soon increases and may be considerable. Ascites is often present. The liver frequently is congested and some connective-tissue hyperplasia may develop and produce a subicteric staining of the skin. Diarrhœa is a common complication, and one that often proves intractable. It may be due to enteritis, but is oftener due to œdema. The discharges are usually copious and are sometimes streaked or mixed with blood. In this stage patients are usually too weak to be about the house, and, for safety, rest is essential.

Uræmia is especially apt to develop. It manifests itself almost constantly in a chronic form, and often ends life by developing acutely. Cardiac thrombosis and consequent embolism may be the immediate cause of death, but it is oftener uræmia or some complicating inflammation. When cardiac weakness develops, the disease completes its course rapidly.

It is most important to prolong each stage of the disease, as danger to life is greater and the course shorter in each successive one. Therefore, as the disease progresses, life is hurried more rapidly to a close.

Diagnosis.—When albuminuria exists, a diagnosis is not difficult. The abundant diuresis, high arterial tension, and cardiac hypertrophy are characteristic of the early stages of the disease. In the last stage the symptoms of cardiac exhaustion and scanty urine, of low specific gravity, are equally distinctive. From secondarily contracted kidney true cirrhosis must be distinguished by its history, and usually by its less abundant albumen and less abundant evidence of fatty change within the kidney.

Treatment.—Important indications for treatment are to avert uræmia and cerebral hæmorrhage, or other common complication. These indications exist in every stage of the disease. There are others that are peculiar to each stage. There is no specific which successfully counteracts the characteristic renal lesion (connective-tissue hyperplasia), although, to limit or cause resolution of it, such drugs as the iodides of sodium or potassium, mercurials, and the double chloride of gold and sodium have been used. I have been disappointed by not obtaining positive good effects from them. Undoubtedly, the first two may prove useful in the rare cases of syphilitic origin. I believe the iodides are useful in renal cirrhosis chiefly by lessening arterial tension and by promoting diuresis, but are of little or no use to prevent the cirrhosis. The chloride of gold and sodium may be tried, for it is thought to be decidedly efficacious by Bartholow, Tyson, and Purdie. Quick results must not be expected of drugs used to meet this indication.

The first step toward successful treatment is to remove the cause of the disease. High living and the use of alcoholics should be forbidden. Exposure to cold, dampness, and sudden thermometric changes may aggravate the renal trouble. They must, therefore, be avoided by change of climate and by careful clothing. In the first and second stages of the malady life may be much prolonged by a permanent residence in equable and balmy climes. Southern California is nearly an ideal climate. Many islands, like the Bermudas and the Sandwich, have similarly equable and balmy air. Florida and many places in the Southern States and on the Mediterranean coasts are also suitable winter climates for those who can make only temporary changes of location. The skin should be protected by wool, so that sudden thermometric changes will not be felt. Food should not be highly spiced, or rich, or contain large amounts of albumen. It must, however, be nutritious and varied. No treatment for this disease is so curative, or, at least, so protecting to life, as a suitable diet. By it the great danger to life—uræmia—may often be averted, and an increase of cirrhosis may be prevented or delayed by limiting the amount of material irritating to the kidneys which is admitted to the blood.

Whenever the daily excretion of urea falls below a normal standard it must be increased by stimulating diuresis, and an accumulation of noxious waste matter must be averted by diminishing the metamorphosis of nitrogenous material in the system. This last may be accomplished by regulating the diet. If uræmia threaten, a non-albuminous diet must temporarily be used, but at other times it need only be simple and free from an excess of meat extractives, such as exist in rich gravies, soups, and game. A modicum of albuminous food may be used if it is simply cooked, and so long as nitrogenous matter is eliminated in normal amounts. Milk is nearly as useful as in chronic parenchymatous nephritis. To stimulate the kidneys when their secreting power is lessened, such salts as the citrate or acetate of potassium or sodium, or carbonate of lithium should be employed. Digitalis and strophanthus may be used, but in the first and second stage very temporarily, for it is not desirable to increase arterial tension when it can be avoided, as it in turn increases the heart's labor and may hasten exhaustion. Strophanthus, in therapeutic doses, contracts the arterioles throughout the body least, and is to be preferred. It is, therefore, evident that close watch must be kept of the quantity of urine and of nitrogenous matter that is being voided. Patients should measure the urine made by them in twenty-four hours, at least twice each week. If it fall below an average, which each patient must determine for himself, a physician's counsel should be sought. However, so great confidence cannot be placed in measurements of the daily quantity of urine voided as in coincident tests of the amount of urea voided; for the nitrogenous elimination does not always fluctuate directly as the quantity of urine does. The former may diminish much more than the latter. Intelligent patients can be easily taught to take the urine's specific gravity, as well as to measure its amounts. The table on page 326, which I

TABLE OF NORMAL QUANTITIES OF URINARY SOLIDS.

Amount of urine voided in twenty-four hours:—																			
In pints.	⅖	⅗	⅘	1	1⅕	1⅗	2+	2½	3	3⅓	4+	4½	5	6	6½	8½	10½	12½	19
In cubic centimetres.	200	300	400	500	600	800	1000	1200	1400	1600	2000	2200	2400	2800	3100	4000	5000	6000	9000
Normal average sp. gr.	1095	1065	1048	1039	1025	1024	1020	1016	1014	1012	1010	1009	1008	1007	1006	1005	1004	1003+	1002
Minimum normal sp. gr. . . .	1065	1043	1032	1026	1021	1016	1013	1010	1009	1008	1006+	1006	1005	1004+	1004	1003	1002+	1002	1001+

have computed, will enable them to determine when they are excreting normal quantities of urinary solids.

This table is calculated in cubic centimetres, and the pints named are approximate equivalents, but sufficiently near for clinical purposes.

In the table the specific gravities represent for the different amounts of urine a normal average of 35 grammes of urea, and a minimum normal of 15.

The diet should be restricted, and the kidneys should be stimulated as soon as the specific gravity falls below the "minimum normal" for the amount of urine made; and the patient should endeavor to keep it at or above the "normal average." It must be remembered that the amount of nitrogenous matter voided varies with the diet. For instance, according to Frank, on a pure animal diet, 51 to 92 grammes of urea will be excreted daily; upon a mixed diet, 36 to 38; upon a vegetable diet, 24 to 28; and upon a non-nitrogenous diet, 16. The amount eliminated daily is greater in men than in women, and in different individuals it varies much. Different observers do not agree as to the normal daily variation, and most of them affirm that about 34 grammes daily is excreted by men, and 25 by women; but 15 to 18 may be said to be the minimum normal. And this is a dangerously small amount, if a patient is on a rich meat diet.

Hot-air baths, which will produce sweating, can be used with advantage to relieve uræmia. Nothing else is more promptly efficacious in chronic uræmia. Laxatives are beneficial by aiding to eliminate vicariously uræmic poisons, and they help to prevent high arterial tension. Drastics may be required for this purpose, but their use cannot be long continued, because they are exhausting. The stools should, however, be kept soft, for if the rectum or intestines are distended with compact fæcal matter the pulse becomes more tense and the labor of the heart is increased.

In the first stage of the disease all unnecessary cardiac labor must be avoided, so that its hypertrophy will not be hastened. To maintain soft stools, moderate doses of Hunyadi Janos water or Rochelle salts are among the best remedies. Sometimes the persistent use of calomel, in small doses ($\frac{1}{8}$ to $\frac{1}{3}$ grain), will keep the bowels moving easily and promote better diuresis, if it is lessened.

In the second stage uræmia and common complications must be avoided, as in the earlier stage. Cerebral apoplexy and other hæmorrhages are most apt to occur in this period of the disease's course. The natural termination of this stage is cardiac weakness. Its postponement is the special indication for treatment. The sclerotic changes which often occur in the arteries of various organs are an important cause of hæmorrhage, but they cannot be much modified by medicine. Brittle arteries will not often break unless they are unusually stretched by blood that is under abnormally high pressure. The cardiac hypertrophy, the renal sclerosis and consequent vascular obstruction, and often the more generalized obstruction which thickening of the arterial walls causes, constantly keep the blood under an abnormally high pressure. There are many other temporary and removable causes of high pressure,—such as undue physical or mental exertion, indigestion, and constipation. Excitement, anxiety, or mental strain of any kind must be avoided. It is not best to require patients to give up business or all mental occupation, for their stimulus is needed if they do not necessitate overexertion, and if the individuals are not too weak. Absolute physical rest is not necessary or desirable, but overexertion, which is exhausting, should not be permitted. Hurried and violent movements, such as running, stair-climbing, rowing, or gymnasium exercises, must be avoided. Occupations should be sought that do not require hurried movements, or heavy lifting or other violent exertion. Gentle exercise, systematically taken, is essential to maintain good muscular strength.

Indigestion, both in this and the first stage, must be promptly treated. It is due to slow digestion, which makes it possible for abnormal fermentation to take place in the undigested food. Digestion may be hastened by administering only such foods as are easily digested, and by avoiding an overloading of the stomach. Food should be taken if necessary four or five times daily instead of three, and in amounts so small that the stomach will not at one time contain more than it can quickly digest. Pepsin is useful in such cases. Slow peristalsis is oftener the cause of indigestion than diminished gastric ferments, for a little of the latter can accomplish much. Peristalsis is best increased by gentle general exercise, or by massage over the bowels, and by frequent deep inhalations of fresh air, which, by better oxygenating and purifying the blood, invigorates the function of every tissue, and also by tonic laxatives like cascara and aloes. Abnormal fermentation can be best prevented by avoiding such foods as are most readily attacked and decomposed into noxious substances,—for example, fats and starchy foods, which are not semi-liquid, granular, or easily made so,—and by administering anti-ferments, such as carbolic acid, resorcin, muriatic acid, etc. These drugs must be used with caution and in small doses, as most of them are eliminated by the kidneys and are irritating to them. A dietetic treatment is to be preferred to a medicinal one, in such cases. Constipation must be prevented, as in the early stage of the disease; or, if it is associated with atonic indigestion, by cascara, or aloes in combination with blue pill or calomel. Drastics may sometimes be needed to relieve uræmia. An occasional gentle purge with salts is useful to lower arterial tension when it increases. Arterial tension must be as closely watched in this stage as must signs of renal incompetence. If arterial tension is perceptibly increasing, as can be most easily demonstrated by the sphygmograph, it should be lessened. This cannot always be permanently accomplished. A temporary lowering of the pressure can be effected with promptness and certainty by the nitrites. A rapid lowering of pressure is usually not so desirable as a prolonged one; therefore, the nitrite of sodium or potassium is the best to use. It can be given in doses of from 1 to 3 decigrammes (2 to 5 grains) three or four times daily. It should not be given oftener than is necessary in order to accomplish the desired reduction in pressure. Its persistent use is often commended, but I do not believe that it is safe to employ it for many days or weeks. It lowers the arterial pressure by lessening the irritability of the vascular muscles and intrinsic nerves. In full doses it simultaneously quickens the heart's action. It lessens the oxygen carrying power of the blood, and diminishes the quantity of urine. The last two modes of action make it contra-indicated for continuous use if there are symptoms of chronic uræmia. It may be temporarily employed to relieve uræmia, for it lessens the excitability of reflex centres. From its action upon the heart and the blood-vessels it is evident that it lessens the heart's strength, and, by diminishing the oxygen carrying power of the blood, would, if continuously employed, hasten fatty degeneration both of the heart and the kidneys. While it is an extremely valuable remedy in renal cirrhosis, it must not be used as a part of routine treatment, but judiciously. Because of the analogous action of the iodides they can be usefully employed in this disease. They do not produce so pronounced an arterial dilatation, but it is more prolonged. They must be given in full doses (0.5 to 1.5 grammes—8 to 20 grains) if possible, and may be used with safety for a long time.

The urine must be watched as closely in this stage as in the earlier. The same remedies may be employed to stimulate the kidneys to functional activity. Digitalis and other vaso-constrictors are contra-indicated. If some allied drug is demanded for a short time, strophanthus is the best. Hot-air baths temporarily increase blood-pressure, but if uræmia is imminent they must be used, and even repeated often. They should, however, be used in this stage of the disease only when it is necessary, and only so long as it is necessary. An abundance of pure, clean air, and frequent deep inhalations of it, are certainly useful to prevent uræmia and fatty degeneration by maintaining good metabolism. Inhalation of oxygen may be tried, for it is believed by many to be of value. It has always disappointed me in such cases.

In the third stage, when cardiac weakness exists, a different class of agents must be used. The chief indication for treatment is to maintain the heart's strength. There is greater danger of uræmia than in the earlier stages. It must be averted by the same regimen and treatment. Arterial pressure is low, and digitalis, convallaria, or strophanthus may be used with impunity, and often are absolutely essential. By them the heart can be, at least temporarily, strengthened. Usually, degeneration as well as muscular exhaustion causes the heart's feeble action. Iron, strychnia, quinia, and other bitters are best calculated to stimulate and maintain healthful tissue-change, and, therefore, to avert dangerous degeneration. They must be used persistently, and usually combined with digitalis. An abundance of fresh air, deep inspirations of it, and very gentle active or passive exercise are also needful. The chances of successfully removing the cardiac weakness are not good, for when it develops the blood is impoverished, the muscular and nervous systems lack vigor and tone, and often the stomach digests food imperfectly. These conditions make it almost impossible to do more than to temporarily strengthen the heart. The diet must usually be poor, both because digestion is not good and in order to avert uræmia. It should, however, be as strengthening and as liberal as is possible under the circumstances. Œdema is a symptom in this stage that requires treatment. Sometimes aspiration of the pleural or pericardial sacs, or incisions at the ankles, are necessary. Usually, reliance is placed upon diuretics, diaphoretics, and cathartics.

In renal cirrhosis opiates are generally contra-indicated, since they lessen the urine and especially the elimination of nitrogenous waste. Their use, therefore, increases the danger of uræmia. It is especially necessary to bear this in mind in treating inflammatory complications of the disease. If opiates are employed the urine must be examined daily, and they must be discontinued if they materially interfere with the renal functions. The persistent and very intractable diarrhœa which sometimes occurs in this disease must be treated chiefly by astringents, such as tannic acid, lead acetate, or nitrate of silver. Œdema or inflammation of the lungs must be treated as under other circumstances.

Prognosis.—It is scarcely necessary to recapitulate the prognosis of this disease. If the malady is recognized in the first stage, and if uræmia or intra-cranial hæmorrhages are escaped, the chance of living from five to ten years is good. It may be possible to prolong life even more than this. If uræmia threaten often, the chances are not good for a long life. The second stage commonly lasts from one to five years, though it may be more prolonged. The third stage rarely extends over a year. To treat the disease successfully the patient must be instructed as to its chronicity, its dangers, how their approach is to be recognized, and regarding the necessity for continuous guidance by a physician.

CHAPTER XXXIV.

SUPPURATIVE NEPHRITIS.

SUPPURATIVE NEPHRITIS is more properly a surgical than a medical disease; but, as a diagnosis is generally demanded of the physician first, it is appropriate to describe it briefly here.

Causes.—Pyogenic matter may gain access to the kidneys through the blood or the urinary channels. When by the former, pyæmia or purulent endocarditis is oftenest the primary disease. Rarely, it complicates other diseases that are accompanied by suppuration, such as dysentery.

Suppurative diseases of the urinary channels, such as urethritis, prostatic suppuration, cystitis, and pyelitis, are oftener its cause. Much less frequently renal suppuration originates by extension of inflammation from the perirenal tissue, or results from penetrating wounds.

Anatomy.—The kidney may be enormously distended and its tissue almost wholly destroyed and replaced by pus. This is rare, except as the result of suppurative pyelitis. Usually, the substance of the kidney is studded with minute abscesses and the organ is very little enlarged. It may retain its normal color, or be mottled. Abscesses are frequently visible through the capsule as yellow spots. Over them the capsule is usually adherent to the cortex. On a cut surface the abscesses, if they are of pyæmic origin, are generally most abundant in the cortex; if they originate from diseases of the urinary channels the pyramids are as apt to be extensively involved as the cortex. The abscesses are often miliary and very numerous. When small they appear as minute yellow spots, usually surrounded by a zone of hyperæmia. Often, when the suppuration arises by extension from the lower urinary channels, the kidney seems striated by lines of pus extending through the pyramids and into the cortex. In these cases the inflammation originates in the renal tubules. When the pyogenic matter reaches the kidney by the arteries, it usually lodges in a small vessel or glomerule. At first a collection of leucocytes and pus-cells marks the site of the abscess. As they accumulate they compress and destroy the neighboring tissue. They may grow large, or coalesce and make cavities the size of a cherry or greater. Both kidneys are frequently affected simultaneously.

Abscesses in the kidney, usually, sooner or later, communicate with the uriniferous tubules, and through them empty pus into the urine. If the ureters are obstructed, as they may be in some cases of pyelitis, pus will not be voided from the body. The pelvis of the kidney will then be converted into a distended sac of pus, which will enlarge by the destruction of the renal substance. In this way very large abscesses may be formed. Instead of finding an exit through the urinary channels, the pus very rarely breaks into the peritoneal cavity, or, after adhesive peritonitis has bound the kidney and intestines together, into the intestines, or externally through the abdominal wall, or by burrowing into the pleura or lung.

Symptoms.—Abscess of the kidney may exist without producing characteristic symptoms. This is most apt to occur in septicæmia. The symptoms, which are of diagnostic value, are pyuria, usually renal pain, hectic fever, and sometimes renal tumor. When there is pyuria, it is necessary to distinguish that which is due to renal suppuration from suppuration of the lower urinary passages. When pus is formed in the kidney, the urine contains a larger proportion of albumen than when pus is formed elsewhere in the urinary tract. Renal tube-casts are often found in the urine, and rarely bits of renal tissue may be discovered in it. The sudden appearance of large amounts of pus in the urine usually signifies the bursting of an abscess into the urinary tract at some point in its course.

Pain in the kidneys may be wanting or may be very slight. It is caused chiefly by stretching the capsule. It is, therefore, commonly inconsiderable, except when the whole kidney is involved, and is greatly distended with pus. When pain exists, it is aching or at least constant and dull. It is usually felt quite as much in front and in the side as in the back. Sometimes the passage of clots, shreds of renal tissue, or calculi, when they are loosened from the kidney, causes renal colic.

The kidney can be felt as a tumor only when it is very much distended. It can then be felt by deep pressure upon the sides of the abdomen. The shape of the kidney can usually be outlined by the palpating hand. When enormously distended it may almost fill one side of the abdomen; when considerably distended, fluctuation may be felt. Usually, the organ is too deeply located to make it possible to elicit this sign. When the kidney is sufficiently distended to be felt, it is usually evenly so. Rarely, the surface is made uneven by projecting abscesses of considerable size. The symptoms of hectic fever are present unless the pus is perfectly drained, spontaneously or artificially.

Prognosis.—The prognosis of suppurating nephritis is unfavorable. Recovery is possible if the abscess or abscesses can be perfectly drained. This is rarely, if ever, accomplished spontaneously. When, as is usual, there are many small abscesses, and especially if they are in both kidneys, drainage by a surgical operation is impossible. Under such circumstances death is almost inevitable. The nature of the primary disease, when there is one, must be considered when the chances of recovery are computed.

Treatment.—Treatment must be supporting and symptomatic. The essential of successful treatment is the removal of the pus and the prevention of its reformation. This may necessitate aspiration, nephrotomy, or nephrectomy. Food should be as generous in amount and variety as the stomach will tolerate and utilize. Renal irritants should be excluded from the dietary, beverages, or medicines of those who suffer from suppuration of the kidneys. Anodynes may be needed to relieve pain. Tonics and hæmatics will be useful if fever is absent and convalescence is beginning. Indigestion may also have to be relieved by appropriate treatment.

RENAL DEGENERATION.

CHAPTER XXXV.

AMYLOID KIDNEY.

Nature and Causes.—This is one of the renal lesions often denominated Bright's disease. It is due to the formation of a chemical substance which, united with the renal tissue, forms a new chemical body that destroys the function and structure of that tissue and replaces it with homogeneous albuminoid matter.

Amyloid kidney is commonly secondary to chronic suppuration, but it has been known to develop in the third week after the onset of acute suppuration. The pus may be formed in any part of the body. Often amyloid kidney accompanies chronic suppuration of the lungs or joints. It may follow syphilis and tuberculosis, even when these diseases do not cause suppuration. Rarely, it has been observed in association with chronic intermittent fever. Oftener it accompanies chronic nephritis, cancer, leucocythæmia, and other cachectic conditions. Very rarely, it occurs without discoverable cause. Amyloid infiltrations are observed oftener in men than in women, and oftenest between the ages of 12 and 50.

Anatomy.—When amyloid changes are not extensive, the kidney does not change in size or appearance. If they are sufficiently extensive to produce appearances that are characteristic, the kidney is large, pale, firmer, and heavier than normal. The surface is smooth, and the capsule is easily removable. The cut surface of the kidney presents the same physical characters. Upon it many glomeruli are visible as gray, opaque dots, and here and there streaking the medulla and cortex similar gray lines are observable. If a solution of iodine is poured over the surface, the gray matter becomes reddish brown and is strongly contrasted with the rest of the tissue, which is yellowish. The kidney may be mottled with yellow or may be diffusely yellowish. The color is the result of fatty degeneration. If a section is examined microscopically, the capillary tufts in the affected glomerule will be found to be partly or wholly homogeneous and semi-translucent. The vessels are swollen and impermeable to the blood. Elsewhere in the medulla the arteries and capillaries are seen to be similarly affected. If amyloid deposits are very numerous they will occur extensively in the vessels and may be in the basement membrane of the tubules. The glomerular capillaries and afferent vessels are first involved. Fatty degeneration of the renal epithelium is associated with amyloid infiltration, but the fatty degenerative and amyloid changes bear no constant ratio to one another. Often the fatty cells are cast off and disintegrate. They partly fill some tubules, or granular matter resulting from them does. Hyaline casts and, less frequently, amyloid casts are observable in the tubules. Sometimes the connective tissue is slightly infiltrated with round-cells. The evidences of fatty degeneration and inflammation may be more noticeable, both clinically and anatomically, than those of amyloid infiltration. In such cases the amyloid disease may escape notice unless it is sought for closely. Other organs, especially the spleen and liver, are apt to be similarly affected.

Symptoms. — As amyloid kidney is usually a secondary disease, its symptoms are associated with those of the primary trouble. Anæmia, emaciation, and weakness are usually due both to the renal and the primary disease. Anasarca is almost invariably present, but exists to a variable degree. It is sometimes great and sometimes slight. It may develop early or late, and is not correlated in degree with the extent of amyloidosis. In the same case the urine often varies greatly in amount. It may be much increased, but is, perhaps, oftener normal or a little diminished. Toward the end of life it is usually greatly diminished. It is peculiarly clear and drops very little sediment when it stands. It is acid. Its specific gravity varies from 1005 to 1015. In the sediment, hyaline and, rarely, amyloid casts are observable. Sometimes granular casts, a few oil-droplets, and granular or fatty epithelial cells are seen. The urine contains a large amount of albumen, although in the rarest cases the latter is absent. Urea is usually diminished in amount, but less so than in nephritis. Although the vessels of the kidneys and other organs are much obstructed, the heart is rarely enlarged. The spleen and liver are commonly much enlarged. The latter can often be felt, beneath the ribs, to be unusually firm and smooth.

Uræmia is rare in simple amyloid kidney. As extensive fatty degeneration, or true chronic nephritis, is frequently associated with amyloid kidney, the characteristic symptoms of the latter may be modified and obscured by the accompanying diseases.

Respiration and bodily temperature are not changed by the renal disease. The appetite and the power to digest vary greatly. Usually, they are diminished, and consequently there is evidence of slow digestion Often there is diarrhœa, which is persistent and not easily controlled. It may be due to intestinal catarrh, ulceration, or amyloid change in the arteries of that organ, or to all these lesions combined.

Death may be due to intercurrent inflammations of serous sacs or lungs, but oftenest to marasmus. The duration of the disease is variable. Its average is one or two years. It is fatal almost with uniformity. Recovery is supposed rarely to have occurred.

Diagnosis.—A diagnosis is frequently difficult or impossible. If there is a good cause for amyloid disease, and if there is an enlarged spleen and liver, abundant albuminuria, a normal or nearly normal quantity of clear urine of low specific gravity, its existence is probable. It can be distinguished from acute nephritis by the small amount of urine which accompanies the latter, its high specific gravity, and its cloudy and reddish color. Blood is very rarely present in the urine from amyloid kidneys. From chronic parenchymatous nephritis, amyloid kidney can be distinguished by the smaller amount of urine, by its greater turbidity, and by its higher specific gravity in the former. From renal cirrhosis it is distinguishable by the larger flow from the former of limpid urine of low specific gravity, containing only traces or small amounts of albumen.

Treatment.—Treatment must be prophylactic, symptomatic, and supporting. Abscesses must be drained; syphilis or intermittent fever must be cured in order to prevent the extension of the amyloid deposits. Other primary diseases must be removed if possible. Œdemas may require removal; indigestion and diarrhœa may need treatment. If the functions of the stomach and of the bowels are much disturbed, it is impossible to properly nourish a patient. When these organs act well the nourishment should be abundant and highly nutritious. The elimination by the kidneys of nitrogenous matter is usually so perfect that meat and eggs can be eaten without danger. Food should be so prepared and so given that digestion will not be overtaxed or impaired. There is no medicinal treatment especially adapted to amyloidosis.

DISORDERS OF THE RENAL PELVIS.

CHAPTER XXXVI.

NEPHROLITHIASIS.

Causes and Symptoms.—When calculi form in the kidney or in its pelvis the condition is called nephrolithiasis. Calculi vary in size from fine, sand-like particles to equal a hen's egg. When small they are usually very numerous. Their number generally varies inversely to their size. They are commonly rounded and smooth, but may be acicular, faceted, or noduled. They are usually composed of uric acid and urates. They may be formed of oxalate or carbonate of lime, or of phosphates, or very rarely of cystin, xanthin, or indigo. Calculi differ in hardness and color according to their composition. Uratic stones are usually brownish or reddish brown. Calcareous calculi are often very hard. Renal stones are frequently of mixed composition. The small ones usually exhibit a crystalline fracture. The larger ones are more granular. They may be laminated. The various layers may have the same or a different composition. Calculi are supposed to form around a nucleus, which may be a crystal or a few epithelial cells, or a clot of mucus, or bacteria. Calculi often form in the kidney's pelvis. Renal sand may be deposited in the tubules or even in the intertubular connective tissue. Upon the cut surface of a kidney it may produce reddish striæ through the medulla and cortex. It will cause a knife to grate as it cuts the organ. Larger calculi may also be deposited in the renal substance, but oftener they are found in the pelvis or, partly imbedded in the kidney, protrude into the pelvis.

The state of the blood, or the condition of metabolism in the kidney or generally, which causes their formation, is not understood. It has been observed that they form oftenest in childhood and old age. They have been seen in infants who died a few days after birth. They occur oftener in men than women. Sedentary habits and high living seem to predispose to them. Occasionally, there seems to be an inherited predisposition to their formation. There are geographical areas where nephrolithiasis is a common disease, and others where it is extremely rare. The use of water that is strongly calcareous predisposes to the formation of lime calculi. They sometimes form in old age, when lime is re-absorbed from bones, and in osteomalacia. Pyelitis, and especially if urinary decomposition occur with it, is often accompanied by the formation of calculi. Gout and the uric-acid diathesis are frequently complicated by nephrolithiasis.

Calculi may exist in the kidney or its pelvis for a long time without causing an appreciable disturbance. Nor does the gravity of the symptoms which they may produce bear any relation to their number or size. They frequently cause pyelitis (see page 350), renal hæmorrhage, inflammation, and colic. When a calculus obstructs permanently a ureter, it may cause hydronephrosis.

Calculous pyelitis is usually diffuse, but may be circumscribed. Ulcers may be caused by it, and lead to perforation and perirenal inflammation, or to communication with the abdominal cavity, or the intestines. *Hæmaturia* is a common symptom. It varies much in severity, but usually is repeated if it happen at all. It often occurs when a patient stands or walks far, and ceases when he is quiet, or occurs only after severe or protracted labor. The blood is usually intimately mixed with the urine. Clots may be passed. Cylindrical ones are casts of the ureters.

Frequently attacks of nausea and even vomiting are the result of reflex irritation by renal calculi. Quite as often frequent urination and vesical tenesmus are produced by them. Inflammation of the kidney is caused by calculi. They are common causes of suppurative, less frequently of chronic, nephritis. If they produce chronic renal inflammation, it is especially apt to be of the interstitial form.

Renal colic and the passage of calculi are the most characteristic symptoms produced by stones in the kidney. The colic often has an abrupt onset. Intense pain develops at once. Less frequently the pain gradually intensifies. If very intense, the symptoms of collapse may develop rapidly. The patient will then be extremely prostrate, almost speechless, his pulse quick, small, and soft, and his skin usually cold and clammy. When the pain is intense it cannot always be located, but is described as a severe abdominal cramp. Oftener it begins over one of the ureters and then becomes diffused over the abdomen. The colic is usually accompanied by pains that extend into the groin and testicle, or into the thigh on the side affected. In milder cases, a steady, teasing pain will be felt in the region of one kidney, or in the loin, which gradually shifts to the region of the ureter, and is felt to move downward toward the bladder. The pains are paroxysmal, or, if constant, become intense, paroxysmally. The pain is undoubtedly due to a spasm of the ureter, and is a true colic. A sufferer from it cannot rest, but walks the floor, or tosses constantly upon a bed. Very nervous persons have been thrown into convulsions by it. Nausea, and frequently vomiting, accompanies these attacks. In many cases, the vesical tenesmus is considerable. The pain may cease suddenly when the stone drops into the bladder. The cause of these symptoms is demonstrated if stones are afterward passed from the bladder, or discoverable in it. If the calculi are sand-like there may be little pain, or the attacks may be mild. Vesical tenesmus is a common symptom even in the mildest cases.

Renal colic must be differentiated from other abdominal colics by the location of the pain, the extension of it to the groin, testicle, or thigh, and usually by simultaneous vesical tenesmus. It is confirmed by discovering the calculus. It is distinguished from hepatic colic by the greater tendency in the latter for the pain to radiate upward toward the heart or shoulder, and by its location just to the right of the epigastrium, and by subsequent jaundice, or by the discovery of bile-stones in the stools.

The symptoms of *hydronephrosis* are, first, those of renal tumor. If the kidney is much distended, it can be felt through the abdominal walls and outlined by a palpating hand. An area of resonance usually separates it from the liver. Dullness and tumors of the latter move with deep respiration, but renal tumors do not. An enlarged kidney can be distinguished from an enlarged spleen, because the latter usually enlarges upward and outward, and may cause a lateral prominence of the lower ribs; but the former enlarges downward and forward, and causes a prominence of the anterior abdominal wall. When the kidney is enlarged the spleen can, by percussion, be outlined in its normal place and demonstrated to be independent of the renal tumor. It can be distinguished from gastric tumor by the movability of the latter when the stomach is more or less distended, and from fæcal tumors by their removal (as is usually possible) by free purgation. From ovarian tumors it must be distinguished by the history of their development upward out of the pelvis. The latter are usually in direct contact with the abdominal wall, and produce an area of complete dullness. Renal tumors, except when very large, are separated by loops of intestine from the abdominal wall, and cause only relative dullness. The tumor produced by hydronephrosis is usually not very great, although, in exceptional cases, it may fill nearly half of the abdomen. It is often somewhat uneven. The prominent parts correspond to the dilated calices. When such a small tumor is discovered, hydronephrosis must be differentiated from solid renal tumors by demonstrating fluctuation of it, and from other fluid tumors, such as abscess and echinococcus. In echinococcus hydatids must be sought in the urine. In abscess the symptoms of hectic fever must be expected. The discomforts which abdominal tumors produce are usually present, especially if the renal tumor is large. They are: abdominal distension, weight and dragging, dyspnœa from pressure upon the diaphragm, or constipation from pressure upon the intestines. Gastric symptoms, such as nausea and vomiting, may arise reflexly. Rarely, a distended renal capsule has been known to rupture and permit the retained fluid to escape into the peritoneum, which uniformly causes acute peritonitis. When hydronephrosis is slight, it may not be discoverable, or may be easily overlooked, unless there is a history of sudden obstruction of a ureter. The anatomical changes consist in a distension of the renal pelvis, a compressing of the pyramids, distension of the calices, a slow atrophy of the renal substance, which is replaced or, in extreme cases, represented by a small amount of connective tissue. It must be remembered that hydronephrosis may be caused in other ways than by lithiasis. The ureters may be congenitally narrow, or compressed by tumors, or twisted; or there may be obstruction to urination at the neck of the bladder or in the urethra. In the latter cases both kidneys are liable to distension. If a hydronephrosis can be diagnosed, it must be treated surgically. Its cause may be removed, or the entire kidney may be taken away. Aspiration for diagnostic purposes is not safe, for peritonitis has resulted from it.

Lithiasis rarely produces a renal tumor by the accumulation of stones in the kidney's pelvis, or by the formation of a very large one that will distend it. When thus impacted, stones produce a hard, nodular tumor of moderate size. Its location and sometimes obstruction of a ureter, or the history of former renal colic, or the passage of sand or gravel, make probable the difficult diagnosis of such lithiasis.

Treatment.—The indications for treatment in nephrolithiasis are the removal of stones and the prevention of their reformation. It has not been demonstrated that stones of any size can be dissolved by medicines which are administered by the mouth. They can only be removed by an operation, and this is only justifiable when the stones are provocative of other renal lesions, such as dangerous renal hæmorrhage, or pyelitis, or hydronephrosis. When, as oftenest happens, stones are passed and the second indication for treatment is the essential one, we may hope for a reasonable degree of success from proper hygiene and medicinal treatment.

If the calculi are composed of uric acid or acid urates, a mixed diet should be prescribed that shall contain a moderate or minimum amount of nitrogenous matter, and exercise and frequent deep inspirations of fresh air should be assured to make metabolism active and complete. Alcoholics should be interdicted, as their steady use prevents perfect tissue-change and promotes the accumulation of waste in the system. Pure water should be taken freely, that the tissues may be well washed and all soluble matter removed. The water should be as free from mineral matters as possible, that its dissolving power may be as great as possible; or it should contain lithium. Lithium and potassium salts can be given in copious draughts of water, as they energize oxidation, and, therefore, make more perfect tissue-change. Therefore, urea will be formed in larger, and uric acid in smaller, amounts. Lithium also unites with uric acid and forms a very soluble compound. In these ways, an overproduction of uric acid will be prevented, and what is formed easily removed with the urine. Vichy, lithia-waters, or such salts as the acetate or citrate of potassium or carbonate or benzoate of lithium, are commonly prescribed. The benzoates are especially useful, for they convert uric acid into soluble hippurates. The benzoate of lithium or sodium can be given in doses of from 0.5 to 1.0 gramme (10 to 15 grains).

The oxalates which may form calculi are chiefly produced from such vegetables as rhubarb, sorrel, tomatoes, tea, spinach, cabbage, and celery. Their use must, therefore, be forbidden. Alkaline diuretics are now useless. The vegetable ones, such as stigmata maidis and uva ursa, are often apparently efficacious. They can be given as fluid extracts, in doses of from 1 to 4 cubic centimetres (¼ to 1 drachm). Water should be taken freely.

Phosphatic precipitates in the kidney can be prevented by maintaining the urine acid. A meat diet will accomplish this in many persons, or acids can be given by the stomach. Dilute nitro-muriatic is oftenest used in doses of 5 to 10 minims, or dilute lactic in doses of from 2 to 4 cubic centimetres (½ to 1 drachm).

When calculi cause colic, the pain must be lessened by opiates, or by anæsthetics, such as chloroform or ether. It must be remembered that, as in other cases of severe pain which may suddenly cease, the anodynes and anæsthetics, if given in very large doses, may produce fatal or dangerous poisoning; for, so long as pain is intense the large doses may not subdue it, although they prove toxic when it ceases. The discomfort which accompanies a mild colic or the passage of sand can often be mitigated by sinapisms and by heat applied externally. If calculi are slow in passing through the ureters, massage can be practiced over them, and atropia and strychnia can be given, as they are supposed to stimulate more vigorous contractions in muscular structures, such as the ureters.

Renal hæmorrhage is best checked by rest, by cold applications over the kidneys, by ergot, gallic acid, and acetate of lead.

CHAPTER XXXVII.

PYELITIS.

Cause.—Pyelitis is an inflammation of the pelvis of the kidney. It may be catarrhal, or purulent, or hæmorrhagic. It is commonly a secondary disease. Occasional cases are met with for which no cause can be assigned. Some of them follow exposure to cold. Infectious diseases, such as typhoid, small-pox, and pyæmia, are often associated with mild catarrhal pyelitis, whose existence is only demonstrated upon the post-mortem table, for the symptoms of the primary disease mask those of pyelitis. It may be provoked by such drugs as cantharides, copaiba, and turpentine. Obstructions in the urinary tract often produce the lesion. For example, compression of the ureters by a pregnant uterus or other abdominal tumor may cause it. In such cases, there is also more or less of hydronephrosis. Oftenest pyelitis arises by extension of inflammation from other parts of the urinary tract, or by irritation from foreign bodies within the pelvis. More or less pyelitis is commonly associated with the various forms of nephritis. Cystitis very often causes pyelitis, and urethritis may do so. The foreign bodies which cause it are usually calculi. Clots and parasites may also produce it.

Anatomy.—The pelvis of the kidney may be acutely or chronically inflamed. When acutely, it may be reddened diffusely or only in patches. The mucous membranes and submucosa become swollen, and mucus, desquamated epithelium, and some round-cells adhere to the surface or mix with the fluid contents of the pelvis. If the inflammation is chronic, the lining of the pelvis is often brownish or grayish in color. The contents may be the same catarrhal products as in acute pyelitis. Hæmorrhage may occur in either acute or chronic pyelitis, and, when it does, often causes extravasation beneath the epithelium and subsequent pigmentation of the mucosa and submucosa. Purulent inflammation is not uncommon. It usually is the result of extension of inflammation from the bladder. In purulent pyelitis the kidneys are usually sooner or later involved. (See page 333.) The whole kidney may be destroyed or transformed into a large abscess. Usually, the pelvis is distended when it suppurates. Instead of diffuse suppuration, ulcers may form and may penetrate the capsule and cause perinephritis or peritonitis.

Symptoms.—A characteristic course cannot be described for pyelitis, because it is usually secondary to other diseases. Acute cases are often unrecognized. Recovery may occur in some.

Frequently, pain is complained of in the region of the kidneys. It is a feeling of tension or aching Often it is associated with pain in the testicle or perineum, or with frequent urination and straining. The passage of calculi or clots may cause renal colic. The urine is usually acid and of normal specific gravity. It contains an excess of mucus and generally some pus; it may contain much of it. Under the microscope, if there is pyelitis, besides pus-cells, triangular and tailed epithelial cells can commonly be seen. The latter are often regarded as quite pathognomonic of pyelitis, but similar cells have occasionally been found in the urine as the result of cystitis. Blood in abundance is rarely observed, except when pyelitis results from calculi. If the kidney is also involved, casts of renal tubules can usually be found in the urine. If fever exist, it is commonly caused by the primary disease, but it may be due to suppurative pyelitis. Emaciation and other symptoms are produced chiefly by the other diseases which pyelitis accompanies, but may also be due to suppurative pyelitis. Headache, delirium, and coma may be due to ammonæmia from the absorption of ammonia from decomposed urine in the bladder or renal pelvis.

Diagnosis.—It is evident that a diagnosis is often impossible. If the existence of calculi or of some other renal affection can be demonstrated that may cause pyelitis, and if, at the same time, in acid urine tailed epithelial cells can be found, a positive diagnosis can be made. It is especially difficult to distinguish pyelitis when there is cystitis.

Treatment.—Treatment must be prophylactic and symptomatic. For example, if cystitis is cured, or if calculi are removed, the pyelitis may be recovered from. Pain must be relieved by opiates. Counter-irritants, cups, or leeches over the kidneys will often relieve the aching. It is especially desirable, when there is slight catarrhal pyelitis, to prevent urinary fermentation within the kidney, or to lessen it, if it exist. For this purpose, such antiseptics as resorcin, acidum salicylicum, and salol are the most useful. They should be given by the mouth, in as full doses as are well borne. Of these, salol is the best tolerated and, perhaps, the most useful. After it is decomposed by the alkaline juices in the duodenum into salicylic and carbolic acids, the latter are eliminated by the kidneys. To limit the formation of pus, oil of sandal-wood, copaiba, and similar preparations are often recommended. They should be used with care, for pyelitis may be produced or aggravated by them. When judiciously employed, they often lessen the amount of pus formed. If suppuration is extensive, surgical treatment may be necessary. The suppurating pelvis may be drained, washed, and dressed as an abscess, or the entire kidney may have to be removed. The last procedure is indicated when the renal capsule is much distended with pus and the renal tissue is mostly destroyed.

In pyelitis food should be simple, nutritious, and free from renal irritants. Therefore, alcoholics and food that is strongly spiced should be forbidden. Milk and milk foods are especially appropriate.

Prognosis.—Mild cases usually recover in one or a few weeks. Suppurative pyelitis that has not involved the kidney extensively may be recovered from. If very chronic, or if suppuration is acute and spreading, the chances of recovery are not great.

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